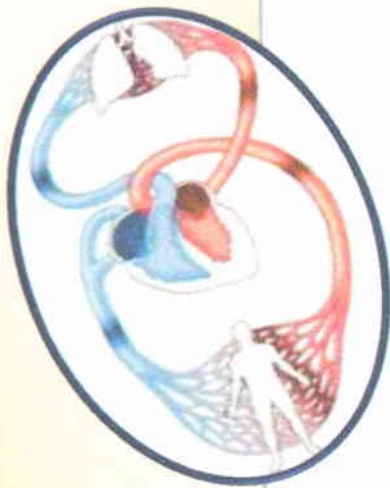
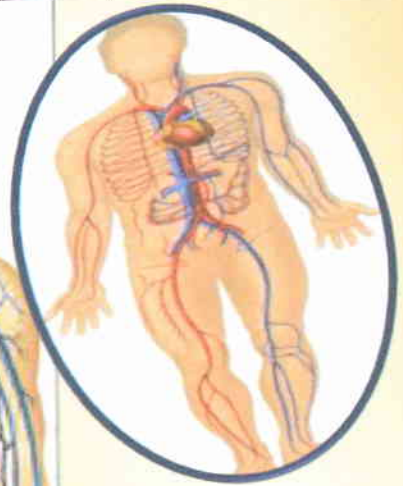
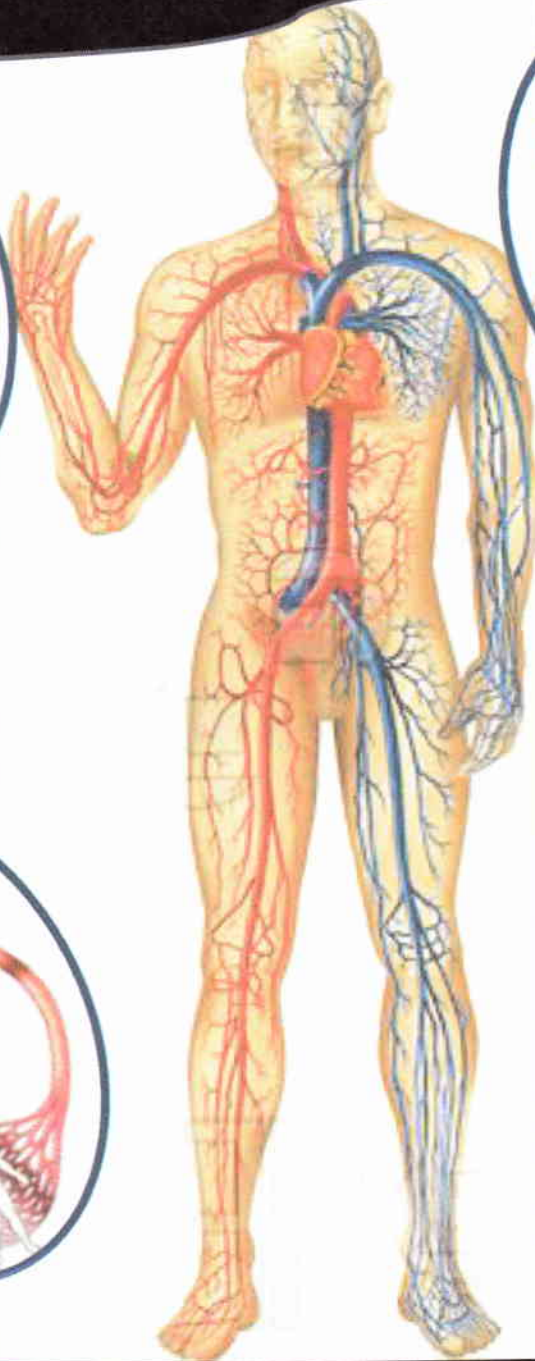


Vascular Surgery

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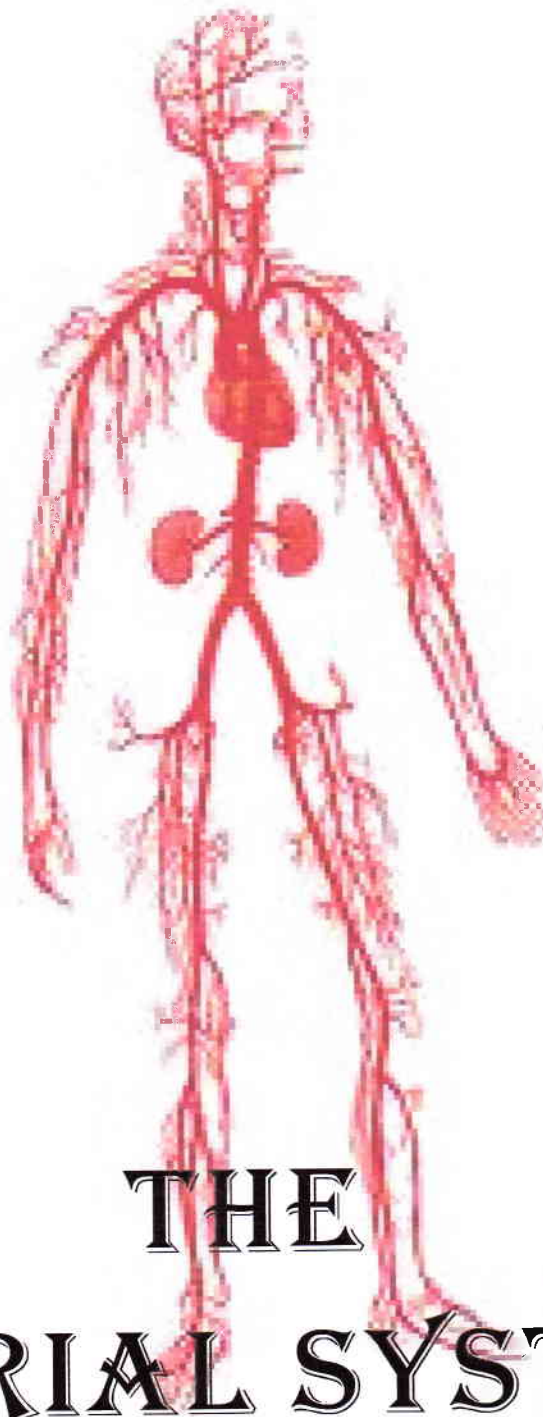
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CHAPETR 1



THE ARTERIAL SYSTEM

Introduction to Arterial Diseases

- **Arteries** are characterized by the presence of collateral circulation, which under normal conditions are collapsed. This circulation opens whenever the blood flow in the main artery is inadequate for tissue requirements.
- **The effect of ischemia depends on:**
 - 1- **Type of artery:**
 - Some arteries have very efficient collateral circulation so that their ligation may not be followed by serious consequences e.g. subclavian artery (due to collateral circulation around the scapula) on the other hand popliteal artery has poor collateral circulation.
 - 2- **Rate of occlusion:**
 - Acute ischemia is much more serious than chronic ischemia as there is no enough time for the collateral circulation to develop.
 - 3- **State of collateral vessels:**
 - Healthy collateral vessels can compensate to some extent the ill effects of ischemia. On the other hand, if the collateral vessels get thrombosed, the effect of ischemia will be disastrous.
 - 4- **General condition of the patient:**
 - The presence of myocardial insufficiency or severe anemia will exacerbate the effects of ischemia.

Acute Ischemia (Acute Arterial Occlusion)

Definition

- Lack of blood flow due to sudden occlusion of a previously patent artery with **no** time for collaterals to open.

- **Pathological definition:**

hypoperfusion of tissues affecting the cellular functions and metabolism

► **Etiology:** TEA.

► **C/P:** 6P

► **Comp.:** Gena, Cat, Volk, Rat

► **Invest.:** art. (duplex, angio), Systems (ECHO)

► **TTT:** Pre, operative, Post. (cause, comp.)

Causes

- 1) Embolism.
- 2) Thrombosis.
- 3) Trauma e.g. catheter, arteriography. Intra-arterial drug injection
- 4) Others (pressure from outside e.g. tourniquet, plaster cast, aortic dissection, phlegmasia Alba dolens).

The most common causes are embolism and vascular injury

Clinical Picture

A. C/P of acute ischemia

- **Onset:**
 - Sudden in embolism.
 - Less dramatic in thrombus.
 - Following trauma in arterial injury.
- **Presentation:**
 - Results in classical picture of **6Ps**:
 1. **Pain:** sudden severe pain, usually felt in the most peripheral part of the limb.
 2. **Paralysis** → The toes cannot be moved in contrast with venous occlusion muscle function is not affected and gradually movement becomes more difficult. Onset of motor paralysis indicates impending gangrene.

3. **Paresthesia** (bad sign) anesthesia often stated to be paresthesia, but in truth, complete loss of sensation in the toes and feet is characteristic (start with loss of touch then progress to loss of deep sensation).
4. **Pallor**:
 - At first leg is marble white and veins are empty.
 - 6-12 hrs later VD occurs and capillaries fill with stagnant deoxygenated blood → mottled appearance
 - Later on capillaries rupture → fixed blue staining of skin (irreversible changes).
5. **Pulselessness** → absent pulsations distal to the site of embolism, but the femoral pulse may be palpable, even thrusting, as distal occlusion results in forceful expansion of the artery with each pressure wave despite the leak of flow.
6. **Progressive coldness of the limb** (Poikilothermia). There is poor capillary refilling



Fixed color changes indicate irreversible stage of acute ischemia → amputation is indicated

B. C/P of the cause

- Embolism: a source of emboli (AF), other system affection (painless hematuria, hemiplegia, or MVO), no history of claudication pain and the condition develop suddenly.
- Thrombosis (intermittent claudication, other system affection (e.g: IHDs).
- History of trauma.

	Embolism	Acute thrombosis on top of atherosclerosis
History	Arrhythmia or recent M.I	Claudication
Source of emboli	Usually present	Absent
Radial pulse	Usually irregular (AF)	Usually regular
Skin colour	White	Dusky
Limb nutrition	Normal	Picture of chronic ischemia eg: Trophic change, rigid blood vessel
Angiography	- Sharp cut-off - Minimal collaterals	- Tapering stenosis - Diffuse atherosclerosis - Extensive collaterals

Classification

- 1- Viable
- 2- Threatened
- 3- Irreversible
 - Fixed color changes (blue staining).
 - Signs caused by muscle necrosis (tense calf, fixed planter flexion, bulging anterior leg compartment).
 - Acute paraplegia may occur in saddle aortic aneurysm.

Moderate limb ischemia

◆ Occurs if:

- 1- Embolus does not cause complete occlusion.
- 2- Pre-existing good collaterals in cases of thrombosis.

◆ The onset is more gradual and the symptoms are less severe.

Pathological consequences

- 1- **Secondary arterial thrombosis:** After circulatory arrest widespread distal intravascular thrombosis occurs. Thrombosis may also affect the artery proximal to the site of obstruction.
- 2- **Compartmental syndrome:** Ischemic muscles get swollen which exaggerates the effect of ischemia. After revascularization further edema of the muscles occurs leading to more interruption of the circulation. Early fasciotomy of the muscles compartments can avoid this problem.
- 3- As a result of stasis of blood in the arterial tree, blood stagnates in the veins draining the limb and **DVT may develop.**

Clinical consequences

- *This depends upon the etiology, site, duration and efficiency of treatment*
 - Complete recovery
 - Complications:
 - Gangrene (wet type)
 - Chronic ischemia
 - Volkmann's ischemic contracture
 - Reperfusion injury:
 - **Definition:** body response following restoration of arterial continuity due to release of high concentration of metabolic products into general circulation (e.g. K^+ , Myoglobin, lactate).
 - **This leads to:**
 - Systemic effect: Crush syndrome, pulmonary edema, ARDS, myocardial dysfunction.
 - Local effect: compartmental syndrome

DD

A-Causes of acute limb pain:

1. **Trauma:**
 - Fracture.
 - Dislocation.
 - Crush injury.
2. **Inflammation:**
 - Rheumatoid arthritis.
 - Ankylosing spondylitis.
3. **Infective:**
 - Cellulitis.
 - Myositis.
 - Osteomyelitis.
 - Septic arthritis.
4. **Degenerative:**
 - Osteoarthritis.
 - Backer's cyst.
5. **Vascular:**
 - Deep venous thrombosis.
 - Acute arterial occlusion.
 - Intermittent claudication.
6. **Neurological:**
 - Sciatica.
 - Peripheral neuropathy.
7. **Metabolic:** Gout.
8. **Others:** cramp.

B-Low flow states.

C-Acute occlusion of popliteal and femoral aneurysm.

D-Aortic dissection.

Investigations (Should be urgent)

A. Site of impaction:

- 1- **Doppler & Duplex scan:** localizes and identifies the presence of embolism and thrombus.
- 2- **Arteriography:** (Mainly preoperative, it is not done in threatened limb).
Its value is in cases of diagnosed acute thrombosis because it provides information that is essential before doing an arterial reconstruction. This information includes:
 - a- Site of occlusion.
 - b- Proximal inflow, i.e., if there is another proximal arterial narrowing.
 - c- Distal run-off, i.e., flow distal to the obstruction.

B. For the cause:

- Echo & ECG → to detect AF.
- X-ray → injuries for fracture

C. For complications:

- 1- High hemoglobin, BUN and creatinine indicate intravascular hypovolemia due to fluid sequestration in the limb.
- 2- Acidosis and raised creatine phosphokinase and WBCs → if muscle necrosis.

Treatment

⇒ Pre-operative Preparations:

- Hospitalization
- **Morphine:** for pain.
- I.V fluids to correct dehydration if present
- Prophylactic **antibiotics.**
 - **Heparin** : Start with a loading dose of 80 IU/kg followed by a maintenance dose of 18 IU/kg/hour. The dose is monitored by checking APTT every 12 hours, which should be maintained at 2.5-3 times the baseline level.
- Care of the **cardiac** condition (O₂, digitalis)

⇒ **Operation:** if ischemia is not so severe that immediate operation is essential, it may be possible to treat either embolus or thrombosis by intra-arterial thrombolysis.

- **Embolism:**
 - Urgent embolectomy (within 6 hours) by Fogerty catheter ±
Fasciotomy (*done in late cases as a prophylaxis to prevent compartmental syndrome*).
 - Followed by anticoagulant.
- **Acute Thrombosis on top of chronic:** thrombolytics followed by revascularization surgery.
- **Arterial injury:**
 - ☞ **First aid:**
 - 1- Control of external bleeding.
 - 2- Correct general condition → resuscitation and monitoring.
 - 3- If due to fracture → reduce fracture 1st & wait for 20 min
 - Pulse return → follow up.
 - Pulse did not return → explore the artery.
 - ☞ **Definitive treatment (within 6 hours), according to type of injury**
 - Spasm → paint the artery with papaverine, if failed → forcible dilatation using Fogerty catheter, if failed → excision & graft.
 - Partial tear → repair with graft.
 - Complete tear → repair if large artery & ligate if small artery.

Treatment

1. **Pre-operative preparation:**
2. **Operative:**
 - a. Embolism.
 - b. Thrombosis.
 - c. Injury.
3. **Post-operative:**
 - a. Cause.
 - b. Complications.

⇒ Post-operative:

- **Treatment of the cause:**
 - Embolism → antiarrhythmic drugs if AF.
 - Exclusion graft if aneurysm.
 - Thrombosis → conservative treatment of chronic ischemia.
- **Treatment of complications:**
 - Gangrene → amputation.
 - Volkmann's ischemic contracture → muscle or tendon transfer.
 - Crush syndrome → IV fluids + alkalinization of urine.

Prognosis: the most important prognostic sign is muscle turgor.

Ischemia is not severe in case of atherosclerosis as pre-existing collaterals → thrombosis with gradual onset and less severe symptoms

In acute limb ischemia based on history and clinical examination → Do not wait for investigation as ischemia time is very precious, hand Doppler examination is enough (absent Doppler signals)

Arterial Embolism

"Embolus is a Greek word that means a plug or stopper"

Definition

As acute ischemia +

- A body that is foreign to the blood stream becomes lodged into a blood vessel causing obstruction.

Etiology

A- Cardiogenic (80%):

From the heart (most common):

- Mitral stenosis with AF [the commonest] (77%)
- Left atrial myxoma- mural thrombus MI
- Subacute bacterial endocarditis.

B- Non-cardiogenic (10%):

1. **From large arteries** (i.e. arterioarterial embolization).
 - Atherosclerosis.
 - Aneurysms.
2. **From veins:** venous thrombi through patent VSD or ASD + Eisenmenger → paradoxical emboli.
3. **Other types:**
 - Air embolism, fat embolism (in bone fracture) therapeutic embolization (in treatment of tumors & bleeding), parasitic embolism (e.g. pulmonary bilharzial and bilharzial Corpulmonale) and amniotic fluid embolism.
 - Malignant cell emboli (hypernephroma and cardiac myxoma).

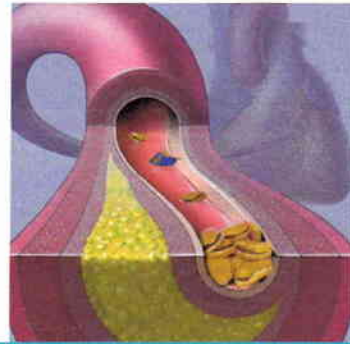
C- Idiopathic (10%).

Pathology

1. An embolus in the arterial tree is usually arrested at the sites of bifurcation of arteries where sudden reduction in the size of the arterial lumen occurs. The lower limb is more commonly affected.
2. **Immediate cut off of the blood supply** → severe ischemia + vasoconstriction of collaterals (reflex due to severe pain).

- ▶ **Def.:** Plug.
- ▶ **Etiology:** Hrt 80%, out of heart 20% (A, V, rare)
- ▶ **Incidence:** Fefe & Popos Be Calm (in order)
- ▶ **C/P:** 6ps
- ▶ **Invest.:** art. Cause, comp.
- ▶ **TTT:** pre, operative (Fogerty embolectomy), post

3. **Muscle necrosis** within 4 -6 hours.
4. **2ry thrombosis** after circulatory arrest widespread distal intravascular thrombosis occurs. Thrombosis may also affect the artery proximal to site of obstruction.
5. **DVT** (as a result of stasis of blood in arterial tree = blood stagnates in veins).
6. **Compartmental syndrome** (if prolonged ischemia): ischemic muscle gets swollen and this in turn exaggerates the effect of ischemia. After revascularization further edema of muscle occurs leading to more interruption of circulation.

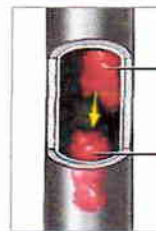


Artery narrowed by atherosclerosis & embolism from heart obstructing the lumen (Acute on top of chronic)

Site

➔ **The commonest sites of arrest of emboli in descending order of frequency are:**

- 1- The bifurcation of common femoral artery into superficial and deep femoral arteries (40%).
- 2- The aortic bifurcation (saddle embolus).
- 3- The bifurcation of popliteal artery.
- 4- The bifurcation of the brachial artery.
- 5- The bifurcation of common carotid artery.



Clot or thrombus

Embolism breaking off from clot

Clinical Picture

- **Presentation differs according to the affected artery.**
 - **Leg:**
 - Sudden onset.
 - As general scheme.
 - **Brain:** the middle cerebral artery (or its branches) is most commonly affected, resulting in major or minor TIA stroke.
- Etc.....



Embolism that has lodged in new location blocking blood

Complications

- See before (acute ischemia).
- Complications are more severe because of little or no collaterals.

DD

1. **Causes of acute limb pain:** see before.
2. **Of the cause** e.g. thrombus.

Investigations

- A. **Site of impaction:**
 - **Doppler U/S, Duplex** → absent blood flow distal to site of occlusion.
 - **Angiography** → block of the main arterial tree.
 - with **no or minimal collaterals**. (unless coexisting atherosclerosis)
- B. **Detection of the cause** → ECG, Echo, U/S if due to aortic aneurysm.
- C. **Detection of complications: muscle necrosis** → ↑TLC, ↑CPK

Treatment

It is a surgical emergency treatment should start as soon as possible.

A. Preoperative Preparation

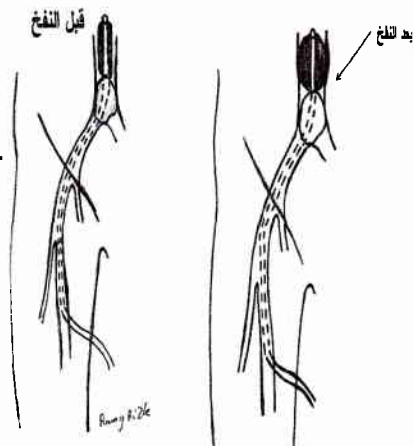
1. **Hospitalization.**
2. **Heparin: (details as in acute ischemia)**
 - To deal with the cardiac problem.
 - To prevent the propagation of the thrombus.

3. **Antibiotics** (prophylactic against infective endocarditis).
4. **Analgesic.**
5. **Care of the cardiac condition:** e.g. digitalis, diuretics, antiarrhythmics ,but the acute ischemia has the priority.

B. Surgical Treatment → Embolectomy.

✦ Operation is done as long as the limb is viable

1. **By Fogerty catheter** (can be done under local , general & epidural anesthesia)
 - For embolism of femoral artery → common femoral transverse arteriotomy
 - For embolism of aortic bifurcation → bilateral femoral arteriotomies
- In both {
- Fogerty catheter inserted till above embolus.
 - Inflate the balloon.
 - Withdraw the catheter.
 - The catheter should also be passed distally to extract any fragments of thrombi
 - Arteriography is done on table to ensure clearance of arterial tree.
 - Inject heparin into blood vessel.
 - Closure by venous patch.
 - Heparin is continued postoperatively according to the cardiac condition of the patient.
 - The source of arterial emboli should be corrected if possible
2. **+/- Fasciotomy** (if prolonged acute ischemia to prevent compartmental \$.).
 3. **Signs of adequate embolectomy:**
 - Pulses are felt during operation.
 - Color & temperature.
 - Reverse bleeding.
 - Intra-operative angiography, Doppler.



C. Postoperative Care

- **Treatment of the cause** e.g:
Anticoagulant (Heparin IV, then continue on oral anticoagulants), antiarrhythmic if A.F, exclusion graft if aneurysm.
- **Treatment of complications**
 - If gangrene → amputation.
 - If Volkmann's contracture → muscle or tendon transfer, if crush syndrome → IV fluids + alkalinization of urine.
 - If MVO → treat, (see GIT).

Insertion of Fogarty catheter in Embolectomy

Complications of treatment by Fogerty's catheter:

- 1- Dissection
- 2- Vessel rupture
- 3- Stripping
- 4- Endothelial damage
- 5- Distal embolization.

Fogerty & his Catheter



Acute Arterial Thrombosis

Definition

As acute ischemia +

- **Thrombosis** is the formation of a clot or thrombus inside a blood vessel, obstructing the flow of blood.

► **Etiology:** old (pt., wall, blood, aneurysm).

► **C/P:** 6ps

► **Invest.:** art. Cause, comp.

► **TTT:** pre, operative (thrombolytics, revascul., amputation), post

Etiology

1. **Age** → old.
2. **Predisposing factors** → **diseased wall:**
 - Arterial narrowing due to atherosclerosis (commonest cause).
 - Endothelial inflammation as in typhoid fever and pneumonia
3. **Precipitating factors** → **stasis & hyperviscosity:**
 - Dehydration (diarrhea).
 - Blood disease (polycythemia).
4. **Others:** aortic aneurysm, traumatic contusion.

Pathology

- It is acute on top of chronic ischemia due to atherosclerosis (fibrofatty streaks, elevated plaques) → collaterals are opened → ischemia is less severe.

Clinical Picture

- **Onset:** acute but less dramatic than embolism.
- **Presentation:** 6Ps.
The limb may be:
 - Viable.
 - Threatened.
 - Irreversible.
- **Past history** of chronic ischemia (intermittent claudications + other systems).

Complications

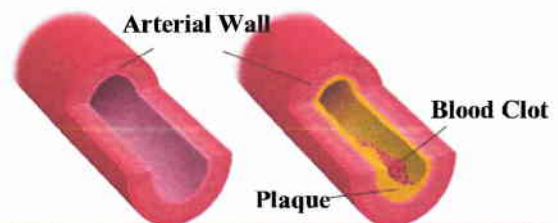
- See before (acute ischemia).

DD

- e.g. embolism + DD of acute limb pain.

Investigations

- Site of impaction** → see before (Doppler & angio) + with appearance of collaterals.
- Detection of the cause** → CBC, lipid profile in blood, FBS, ECG
- Detection of complications:** muscle necrosis → ↑TLC, ↑CPK.



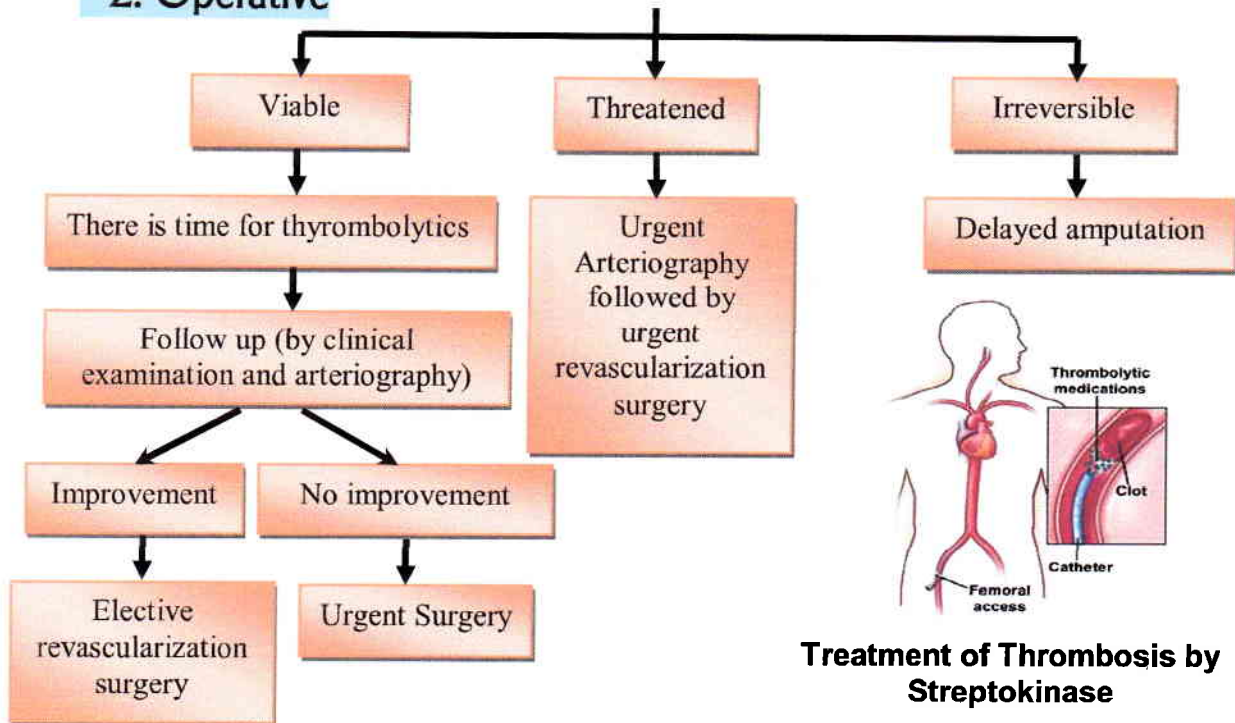
Artery Narrowed by Atherosclerosis

Treatment

1. Preoperative Preparation

- Correct general condition → e.g. dehydration.

2. Operative



Thrombolytics

- Streptokinase is inexpensive but may cause anaphylaxis or be inactivated by antibodies.
- Urokinase is a direct plasminogen activator but is very expensive
- **TPA** (tissue plasminogen activator) results in faster lysis. It is very expensive.

3. Postoperative

- **Treatment of the cause:** conservative TTT of chronic ischemia.
- **Treatment of complications:** (as embolism).

- Thrombectomy by **Fogerty's catheter** is contraindicated because the intima is atherosclerotic, so if it is used, eversion of the intima can occur because thrombosis occurs on diseased intima so, it is friable so we can't remove thrombus but we can dissolve it in contrast to embolism, the intima is healthy so we can remove it.

Peripheral Arterial Injury

Etiology

- **Open injury** (high velocity e.g. missiles or low velocity e.g. knife).
- **Closed injuries** e.g. road traffic accidents. (direct injury to major vessel or by fractures or dislocations)
- **Iatrogenic** e.g. cardiac catheterization, angiography, dialysis.
- **Intra-arterial drug injection.**

Rule of 2

- ▶ With tear, without tear.....2
- ▶ Hard signs, soft signs.....2
- ▶ **Comp.:** CB4 + hematoma.....2
- ▶ **Invest.:** with invest, without invest.....2
- ▶ **TTT:** 1ry then 2ry survey then with tear.....without tear.....2 x 2

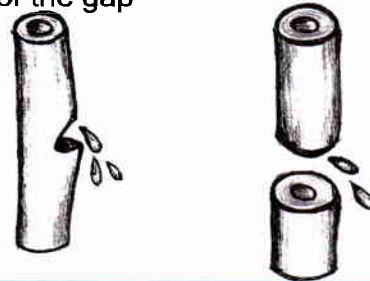
Incidence

- **Peripheral arterial injury accounts for 80% of all the cases of vascular trauma**

Types

⇒ With Tear

- Tear: may be
 - a) **Complete** → ends retract, constrict, thrombose → present with ischemia rather than hemorrhage.
 - b) **Incomplete** → ends retract leading to widening of the gap → usually present with hemorrhage
 - If such bleeding remains within tissue → pulsating hematoma will develop → **false aneurysm**
 - Less commonly if concomitant injury to adjacent artery and vein may lead to **A-V fistula**.



⇒ Without Tear

- **Spasm:**
 - Present by ischemia. Arterial spasm should not be diagnosed except after exploration of the artery to exclude contusion and thrombosis.
- Arterial contusion and thrombosis is the commonest type in cases of closed injuries.
 - Patients present by ischemia.

Incomplete injury

Complete injury

Clinical Picture

- **History of the cause:** trauma, bleeding (if external).
- **General examination** → shock, associated injuries.
- **Local examinations** are the bases for diagnosis
 - **Hard signs:** these are sure signs of arterial injury.
 1. External bleeding (pulsatile hemorrhage).
 2. Loss of distal pulsations.
 3. 6Ps.
 4. Pulsating or expanding hematoma.
 5. Palpable thrill or audible bruit heard at or distal to area of injury.
 - **Soft signs:** these are less specific signs
 1. Small or moderate sized. hematoma that is not pulsating and not expanding.
 2. Proximity of penetrating wound to a major vascular structure.
 3. Adjacent nerve injury producing neurological deficit.



Acute LL ischemia due to arterial injury. NOTE severe pallor

4. History of pre hospital hemorrhage that has stopped or presentation with shock that cannot be explained by other injuries.
5. Delayed capillary refilling time.

Closed injury is more dangerous due to missed diagnosis.

In the upper limb

- Threatening ischemia is caused only by injury to brachial artery proximal to the origin of profunda brachii or occlusion of both ulnar and radial arteries, this is due to extensive collateral circulation and small muscle mass

Complications of arterial injury

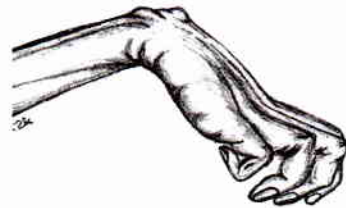
1. Acute ischemia.

2. Hematoma.

- Injury to artery → pulsating hematoma → false traumatic aneurysm (the wall is fibrous tissue & not normal vessel wall).
- Injury to artery & vein → A-V fistula → varicose veins & ischemia).

3. Muscle affection (according to collaterals)

- If good collaterals:
 - Volkmann's ischemic contracture
 - Acute on top of chronic ischemia due to collaterals as the cut of blood supply is partial.
 - Ischemic fibrosis of the muscles.
 - trial to extend fingers passively is associated with flexion of the wrist.
- If bad collaterals → wet gangrene.



NB: Flexion of the wrist → ↓ clawing.

Investigations

- In patient with hard signs: no need for investigations, proceed to urgent exploration.
- In patient with soft signs: urgent investigations
 - Angiography (most accurate). A traumatic A-V fistula shows early venous filling
 - Doppler.
 - X-ray for associated fractures.
 - If soft sign is shock → no investigations should be done, resuscitation & monitoring is the first priority. Arteriography can be done in operating room

Treatment

It could be isolated injury or part of polytraumatized patient, so TTT is

1. ABCDE (primary survey)

Airway

- Prevent backward falling of the tongue by oropharyngeal airway
- Suck any secretion or blood in the mouth with proper oxygenation.
- In comatose patient → endotracheal tube & mechanical ventilation

Breathing

- Early detection & correction of pneumothorax, cardiac tamponade & hemothorax

Circulation

- Control bleeding by local compression or tourniquet.
- TTT of shock either hypovolemic, cardiac or neurogenic.
- Disability
- Any fracture should be splinted to relieve the pain

Exposure

2. Secondary survey

- Head to toe examination of undressed & stable patient.
- History of any (AMPLE H/O).
- Urgent investigation after basic life support.

3. Trauma could be open or closed

❖ Open:

- ☞ Irrigate wound with saline.
- ☞ Wound debridement.

- Skin: excise 1 to 2 cm from edges.
- Fascia: open tense fascia.
- Muscle: excise dead muscle (bluish in color, doesn't contract on pinching, doesn't bleed on cut).
- Nerve: mark with black silk for delayed repair.
- Blood vessel: **see later.**

❖ Closed:

- ☞ If *due to fracture* → reduce fracture 1st & wait for 20 min
 - Pulse return → follow up.
 - Pulse did not return → explore the artery.

- ☞ *On Exploration* → Deal according to type of injury.

1. **Spasm** → paint the artery with papaverine,
if failed → forcible dilatation using Fogarty catheter,
if failed → excision & graft.

2. **Partial tear** → repair with

- If less than 1/2 circumference → Prolene sutures → Saphenous v patch.
- If more than 1/2 circumference → cut it and deal with it as complete tear

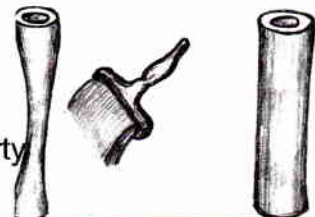
3. **Complete tear**

- Repaired by direct end to end anastomosis.

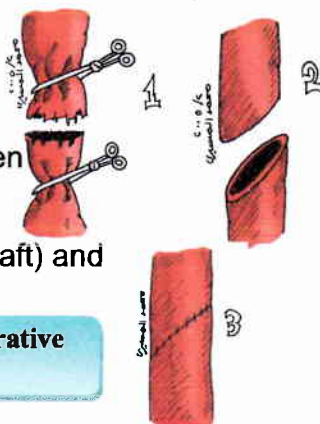
4. **Contusion**: excision of the whole segment and reconstruction with interposition graft (vein or less often synthetic).

Then

Local heparin, fasciotomy, adequate skin covering (graft) and prophylactic antibiotics.



In spasm, paint the artery with Papaverine



Repair in oblique line

N.B. In case of post-interventional vascular complications, post-operative angiography should be done

Complications of vascular repair

1- Immediate postoperative:

- Thrombosis of repaired vessels
- Reperfusion injury

2- Early postoperative:

- Venous thrombosis
- Infection in the area of vascular reconstruction
- Disruption of suture line and hemorrhage

3- Late complications:

- Aneurysmal changes in the vein graft use
- Chronic venous insufficiency after venous thrombosis or ligation.

Intra-Arterial Drug Injection

- The most commonly punctured arteries are the brachial and radial arteries.
- **Clinical features:**
 - 1- Burning discomfort extending from the point of injection to the tips of fingers immediately after injection. This followed by severe pain and blanching.
 - 2- Edema develops rapidly and may be severe.
 - 3- Coldness and cyanosis.
 - 4- The distal pulses are often normal.
 - 5- Digital gangrene and less commonly total extremity gangrene.
 - 6- Arteriography should not be done.

Treatment

- **Treatment should start immediately.**
 - 1- Heparin 10000 IU I.V followed by constant infusion.
 - 2- Dexamethazone 4mg I.V every 6 hours.
 - 3- Low molecular weight dextran minimizes platelet aggregation.
 - 4- Strong analgesics.
 - 5- Limb elevation to decrease edema.
 - 6- Fasciotomy is frequently needed due to chemical myopathy.

Chronic Peripheral Arterial Diseases (PAD)

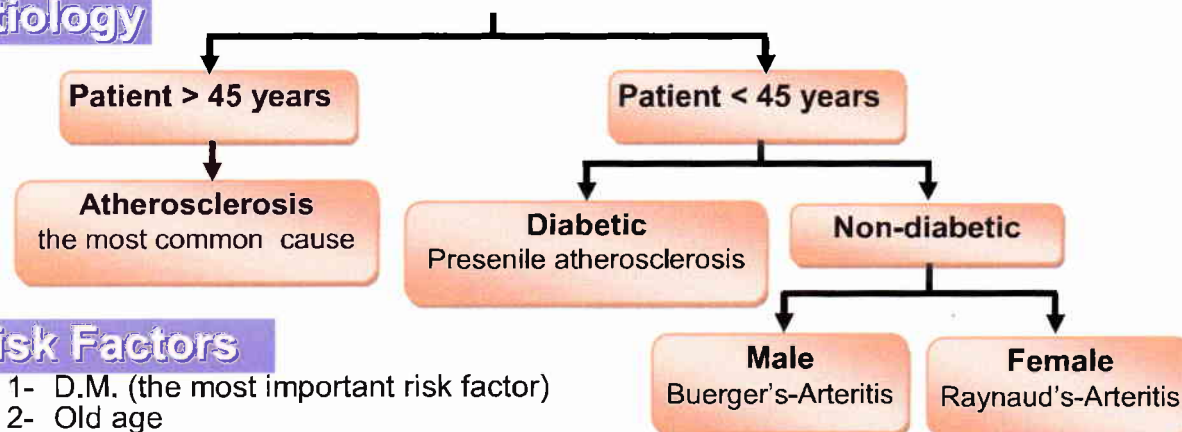
- **In chronic peripheral arterial diseases, patient may be:**
 - 1-Asymptomatic:
 - 2-Claudicant
 - 3-Presented with critical limb ischemia (CLI)

- ▶ **Etiology:** (High).....(BP, Sugar, Age...)
- ▶ **C/P:** Asymptomatic, claudicant, critical
- ▶ **Invest.:** art., other systems.
- ▶ **TTT:** BMT + Revascul. + Amp.

Incidence

- Asymptomatic peripheral arterial diseases (PDA) → 10% of elderly population)
- Intermittent claudication → 5% of elderly population
- CLI → 0.05 % of elderly population

Etiology



Risk Factors

- 1- D.M. (the most important risk factor)
- 2- Old age
- 3- Smoking → 3 times ↑ the incidence of PDA
- 4- Hypertension → BP > 160 / 95 mmHg → ↑ 4 times incidence of PDA
- 5- Dyslipidemia
- 6- Hyperhomocystenemia

Pathology

Atherosclerosis	Buerger's (Thrombangitis obliterans)
It is a systemic disease characterized by deposition of fibro-fatty streaks under the intima of the arteries forming atheroma.	It is an occlusive disease of the small and medium sized arteries, thrombophlebitis of the superficial and deep veins and Raynaud's phenomenon.
▪ Degenerative diseases of the arterial wall. ▪ Characterized by deposition of lipids in the vessel wall → atheroma "elevated yellow streak on the intima" ▪ Occurs commonly adjacent to arterial bifurcation at the origin of major vessels and at the sites where arteries pass beneath or through a fascial sling.	▪ Inflammatory disease of the neurovascular bundle in all 3 layers of the arterial wall + perivascular space (veins & nerves). ▪ The lumen of the artery is obstructed by a thrombus, which becomes organized. ▪ Affection of the nerves by the inflammatory process and the early block of arteries explain the severe pain that is present
▪ Progressive in course. ▪ Affecting proximal vessels. ▪ Usually Symmetrical. ▪ Neuritis is late → complication. ▪ Sites: in descending order of frequency: the coronaries, carotids, lower limb, renal, SMA, celiac and finally arteries of upper limb.	▪ Fluctuating in course. ▪ Affecting distal vessels. ▪ Asymmetrical ▪ Neuritis is early

Clinical Picture of PAD

- 1- Asymptomatic
- 2- Claudicant patient
- 3- CLI

1- Patient may be Asymptomatic

2- Claudicant Patient

a- Type of patient:

Atherosclerosis	Buerger's (Thrmobangitis obliterans)
Male >50 year with risk factors e.g.: - DM. - Obesity. - HTN. - Hypercholesterolemia.	Male 20 -40 year, heavy smoker.

b- Presentation:

i. **Pain:** (it may take one of the following forms)a) **Intermittent claudication:**

- Site → depends on the level of obstruction (see later).
- Cause: is due to stimulation of nerve endings in the muscle by accumulated metabolites during exercise, which will be washed out during rest.
- Character → cramp-like
- Comes on → walking & exercise.
- Relieved by → standing still.

- Claudication distance →

- In early stages of the disease, patient gets the pain after walking for a certain distance (claudication distance) but he can continue his walk. Later on, pain gets worse and forces the patient to stop for few minutes, as the disease progress:

→ Claudication distance → gradually shortens.

→ Period of the rest → gradually increase.

ii. Associated other systems:

- **CVS** → heart failure, angina pectoris & MI.
- **CNS** → TIA and stroke (loss of memory, fainting, blindness or hemiparesis).
- **Kidney** (atherosclerotic kidney) → pain, hematuria, hypertension and renal failure.
- **Genital** → impotence in males.
(**Leriche syndrome = aortobi-iliac obstruction**).
- **GIT** → cramping pain in the abdomen in relation to meal.
(Post-cibal abdominal angina).

Signs:

i. General examination:

- **Chest E** → chest infection.
→ Heart → heart failure, hyperdynamic circulation due to AVF and anemia.
- **Vital signs** → pulse : big pulse volume → AVF and anemia.
→bruit: → systolic → aneurysm
→continuous → AVF.
→ state of vessel wall: rigid in atherosclerosis.

ii. Local examination (limb in layers):

1. Skin:

- Cold sensation: ischemic limb is colder than the other limb but an ischemic limb tends to equilibrate with the temperature of its surrounding and may feel quite warm under the bed clothes.
- Trophic changes:
 - Loss of hair (1st trophic change).
 - Thin, atrophic, stretched, dry skin.
- Color changes
 - Pale → bluish or red → blackish (CLI).
 - It is affected by change of position as affected legs blanch on elevation and develop a purple discoloration on dependency.



- Pallor is due to decrease blood flow to the skin.
- Cyanosis and redness are due to stagnation of blood in markedly dilated capillaries under the effect of accumulated vasodilator metabolites. The color at first red then later on becomes bluish due to extraction of O₂ by tissues.

2. Nails → brittle.

3. Subcutaneous tissues:

- ↓ Limb circumference.
- Thin tapering toes.

4. Muscles:

- Atrophy → weakness.
- Fibrosis.

5. Nerves:

- **Sensory** → paraesthesia, hypo or hyperaesthesia (proprioception is the first to be lost).
- **Motor** → weakness (fine movements are the first to be lost).
- **Reflexes** → weak ankle (S_{1,2}) & knee reflexes (L_{3,4}).

6. Veins:

- Slow venous refilling.
- DVT (2ry to stasis).
- Migrating superficial thrombophlebitis → in Buerger's only.

7. Arteries:

Palpation:

- Weak (in presence of good collateral) or absent peripheral pulsations according to level.
 - Dorsalis Pedis a.
 - Anterior tibial a.
 - Peroneal a.
 - Posterior tibial a.
- Expansile pulsation → may indicate an aneurysm.
- Stenosis or occlusion with a well developed collateral circulation may allow distal pulses to be normal in palpation. In this case, the sign of disappearing pulse may be useful.

8. Bone → osteoporosis.**9. Special tests**

Capillary refilling test, Buerger angle, Harvey's venous refilling time
Disappearing pulse test (see oral questions)

3- Critical Limb Ischemia

- a) Type of patient: As claudicant
b) Presented with: one of 4 diagnostic criteria

I. Rest Pain:

- ▣ **Site**: never comes above the ankle
- ▣ **Cause**: ischemic neuritis
- ▣ **Character**: severe pain awakes the patient from sleep
- ▣ **Comes on**: rest and characteristically it worse at night
- ▣ **Relieved by**: hanging the foot out of bed and by sleeping on a chair
- ▣ **Exacerbated by**: lying down and elevation of the foot

II. Ulcer resistant for healing:

- ▣ **Site**: between the toes, in the dorsum of the foot or around the malleoli
- ▣ **Edge**: Punched out
- ▣ **Margin**: blackened and mummified
- ▣ **Floor**: granulation or pus
- ▣ The ulcer is extremely tender & its base is difficult to be palpated

III. Gangrene: (usually dry unless with HF or infection).**IV. Absence of palpable pulse:****Clinical staging of chronic limb ischemia**

- **Stage I**: asymptomatic.
- **Stage II**: intermittent claudication.
- **Stage III**: rest pain.
- **Stage IV**: ulceration or gangrene.

⇒ Capillary refilling test:

- If doctor presses over tip of the toe, it becomes pale. Once the pressure is released the color returns. In an ischemic limb, the return of color is slow and is called sluggish capillary circulation.

⇒ Harvey's venous refilling time:

- With the patient supine, the limb is elevated to right angle until all veins empty. It is then brought down to the horizontal position. Normally the veins fill in 10-15 seconds.

→ Slow refilling → arterial insufficiency.

→ Fast refilling and varicose veins → AVF.

⇒ Disappearing pulse test:

- In patient with chronic ischemia, dorsalis pedis pulse disappears after exercise.
- Arterial occlusion + good collaterals → normal distal pulse → exercise up to claudication → decrease pulse → 1-2 minutes they reappear.
- The explanation is that exercise produces vasodilatation below the obstructing lesion and the increase in arterial inflow is less than the increase in the vascular space so the pressure falls and the pulse disappear.

Differential diagnosis

► Other causes of chronic pain in the lower limb:

- 1- Sciatica
- 2- Varicose veins
- 3- Flat foot
- 4- Peripheral neuritis

Investigations of PAD

⇒ Investigations for diagnosis:

- a. Doppler, Duplex.
- b. Angiography.
- c. MRA.

⇒ Investigations for associated systems.

A. Diagnosis:

1- Doppler flow study

- d- Stenosed or occluded segments can be detected.
- e- Collateral refilling of the post-occluded or post-stenosed arteries.
- f- Measurement of the ankle/brachial(A/B) index:
 - Normally A/B index is 1-1.2
 - Less than 0.9 denotes ischemia
 - Less than 0.7 indicates severe ischemia
 - Less than 0.3 indicates impending gangrene



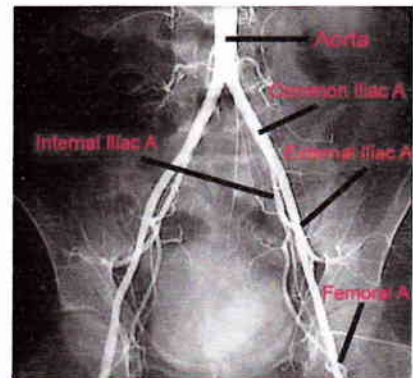
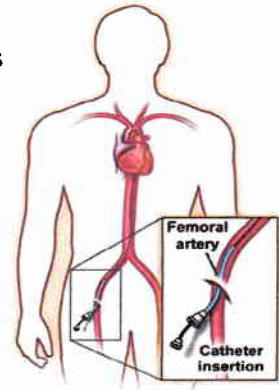
Doppler U/S

2- Duplex scanning

- Visualizes the arteries and detects the areas of stenosis and block

3- Arteriography

- This invasive method is done only if revascularization surgery is intended.
- **Techniques:**
 - a- Direct femoral arteriography: this technique is losing popularity nowadays in favour of more informative aortography.
 - b- Translumbal aortography: this is performed only if both femoral pulses are not palpable.
 - c- Transaxillary aortography: performed only if the whole distal aorta is occluded.
- **Arteriography provides the following information:**
 - a- Exact site of arterial block
 - b- State of the proximal arteries.
 - c- Distal run-off.
- **Complications:**
 - 1- Neurological deficit
 - 2- Hemorrhage
 - 3- Diminished pulse
 - 4- Pulsatile mass (hematoma – false aneurysm)
 - 5- Infection (at puncture site)
 - 6- Allergic reaction to the contrast media
 - 7- Thrombosis of the punctured vessel
 - 8- Death < 0.05 %.



Normal aortography

4- Digital subtraction arteriography (DSA)

- The contrast material is injected I.V

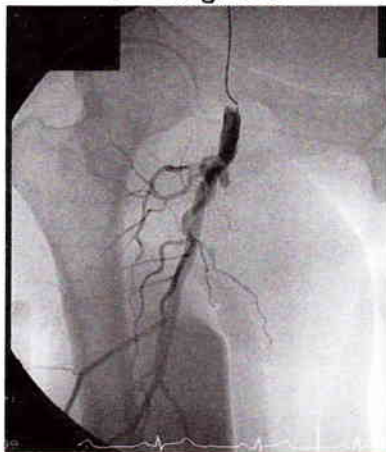
5- MRA (Magnetic Resonance angiography):

a. Advantages:

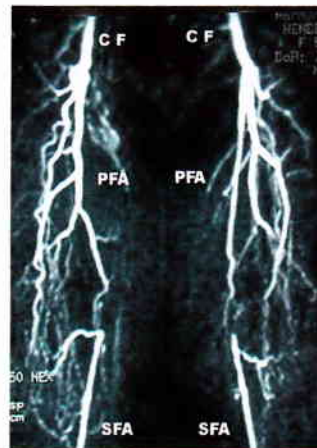
- 1. Non-invasive.
- 2. Same accuracy as arteriography.

b. Disadvantages:

- 1- Expensive.
- 2- New technique & still not experienced enough.
- 2- Diagnostic not Therapeutic



Digital subtraction arteriography of the right leg of the same patient showing a stump in the take off of the superficial femoral artery



Magnetic resonance arteriography showing bilateral total superficial femoral occlusions in a patient with a non-healing ulcer of the right lower extremity. (CFA = Common femoral artery, PFA = Profunda femoral artery, SFA = Distal superficial femoral artery filling from collateral flow from the profunda femoral artery with no visible proximal run-off)

6- Computerized tomography (CT angiography)

N.B. MRA and CT are gradually replacing DSA.

N.B.

- ☞ *In claudicant patient starts the investigation with non-invasive vascular laboratory test and then proceed to one of the imaging tests only if non-invasive test results are insignificant*
- ☞ *In CLI do the non-invasive vascular lab test together with one of the imaging tests according to the clinical situation*

B. Other system evaluation (very important):

- Blood picture to detect anemia or polycythemia
- Blood sugar
- Serum lipids
- Serum creatinine
- ECG and fundus examination.

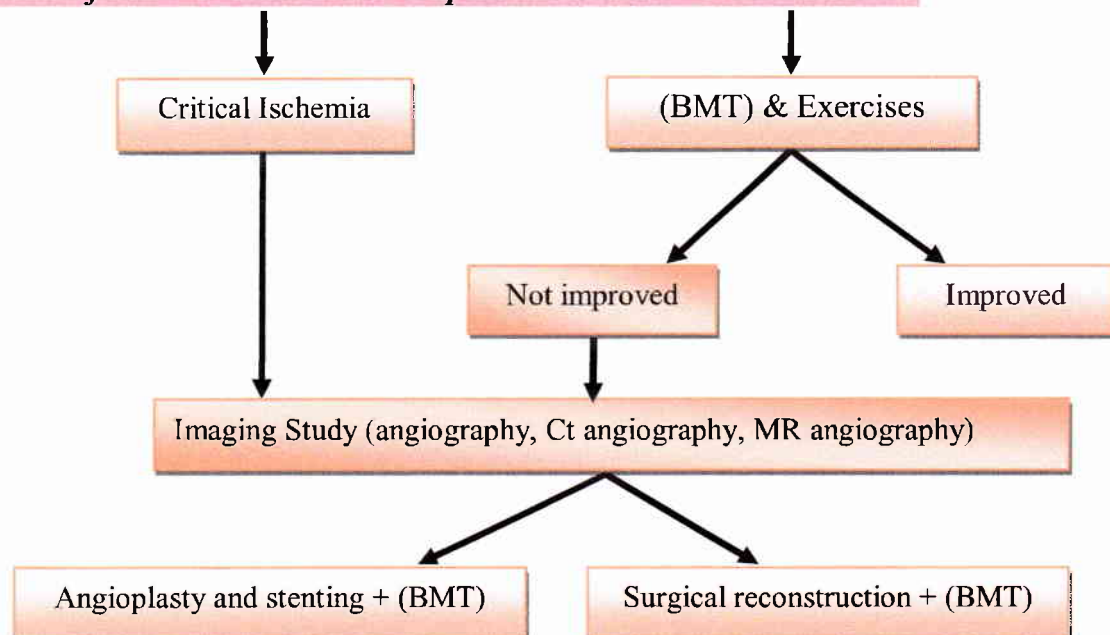
Treatment

Conservative Treatment

Treatment is mainly conservative

Spontaneous improvement occurs in many patients over the 1st 6 months after an occlusive episode as collateral vessels develop.

Symptoms of PAD → Clinical and Duplex assessment → Claudication



A- Best Medical Treatment (BMT):

1- Risk factors modification:

- a- Smoking cessation
- b- Proper control of DM
- c- Control of blood pressure
- d- Lipid lower drugs (statins = HMG-CoA reductase inhibitors)
- e- Vitamin B6 and folate may decrease levels of homocysteine

2- Anti-platelets:

- a- Aspirin 75-150 mg daily
- b- Clopidogrel (Plavix) 75 mg daily, for patients who are intolerant to aspirin

3- Vaso-active drugs (may have a role in intermittent claudication)

- a- Naftidrifuryl (Praxilane)
- b- Cilostazol (Pletal)

4- Care of foot (especially in diabetics)

- a- Avoid cutting angles of the nails, avoid tight shoes.
- b- Good hygiene & dryness of foot.

5- Exercise training:

Walking to near maximum pain producing distance 3 times per week improves the walking distance for patient with intermittent claudication

B-Endovascular Surgery**A. Percutaneous Transluminal Angioplasty (success rate 95%)****Indications (as endarterectomy)**

- Short segment affection in a big vessel (2cm or less).
- Non-calcified
- Not done in occlusion below knee level. - Good outflow

Complications:

- Recurrence.
- Hematoma.
- A-V fistula.

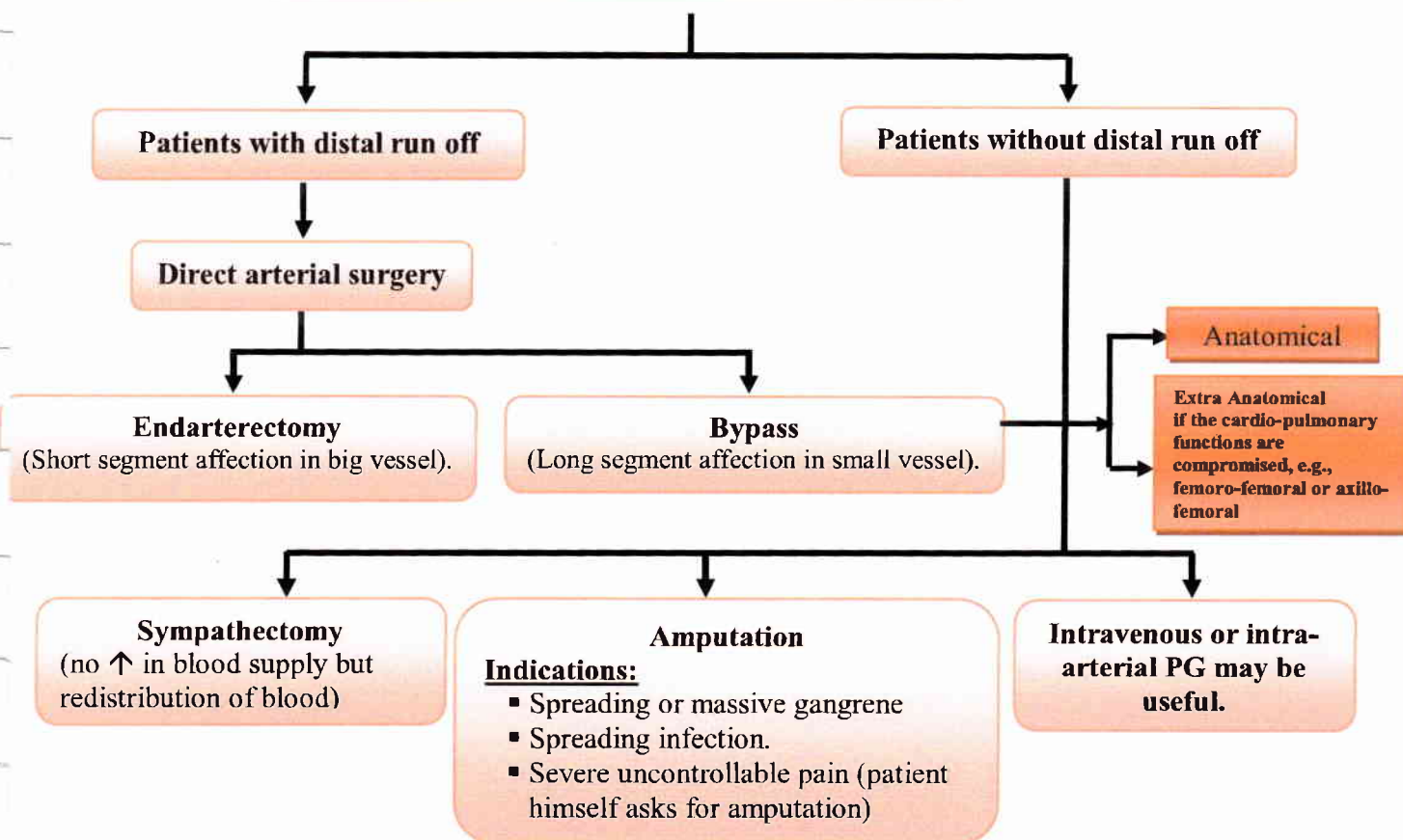
B. Intraluminal Stent: (after balloon angioplasty, stent can be inserted to prevent the elastic recoil of the arterial wall and keep the lumen patent)**C- Surgical Treatment**

(Surgery in this case aims in saving the limb and thus called limb salvage surgery)

⇒ Indications of surgery (=late ischemia)

1. Starting gangrene (to avoid spread of gangrene).
2. Pregangrene.
3. Severe claudication pain interfering with patient's work (differs according to each patient).
4. Ulcers resistant for healing.
5. Long segment occlusion > 12 cm

⇒ **Surgical techniques (according to angiography):**



OPERATIVE DETAILS

► Direct arterial surgery

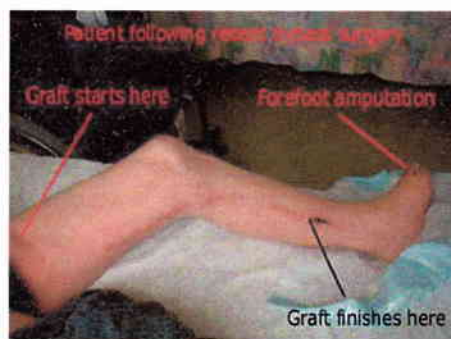
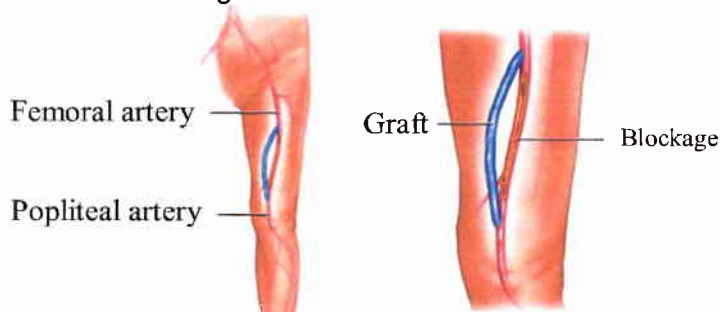
1- Thromboendarterectomy: This means removal of the thrombus, intima and the inner media.

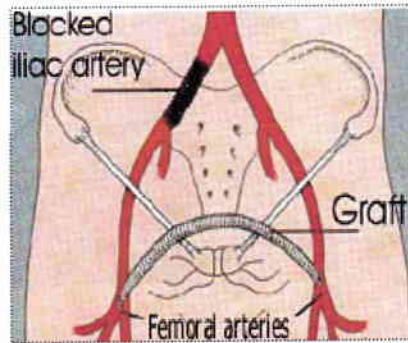
2- Bypass surgery:

a. For aorto-iliac disease: done using synthetic graft made of Dacron or PTFE.

b. For superficial femoral artery block: The standard operation is femoro-popliteal bypass using the saphenous vein.

- To overcome the valves of long saphenous vein one of two techniques is used:
 - Reversed saphenous venous graft.
 - In situ saphenous vein graft. The valves are destroyed and the tributaries are ligated.





3- Lumbar sympathectomy:

➤ Indications:

- Where direct arterial surgery is not technically applicable i.e. cases of distal occlusions with no run off.

1. **Buerger's disease:** vaso-occlusive disease → sympathectomy only to improve collaterals.
2. **Raynaud's disease:** vaso-spastic disease → sympathectomy gives better results than Buerger's.

➤ Contraindications:

1. Atherosclerosis **except with tissue loss (ulcer resistant for healing)** → improves collaterals → improves healing (as it shifts blood to skin) (done in combination with direct arterial surgery).
2. Diabetics, as the patient is already sympathectomized.

➤ Types:

1. Ischemia of UL → **Cervico-dorsal sympathectomy.**
 - T2, 3, 4 are transected.
 - T1 (or stellate ganglion) is left to avoid Homer's syndrome.
2. Ischemia of LL → **Lumbar sympathectomy.**
 - L2, 3, 4 are transected.
 - L1 is left to avoid retrograde ejaculation.

➤ Disadvantages:

- **Redistribution of blood** → ↑ blood supply to skin & ↓ blood supply to muscles → **worsen of claudication pain** (steal phenomenon).

- **Recurrence:** after few years due to → hypersensitivity to circulating catecholamines = **denervation hypersensitivity.**

➤ Technique:

▪ Cervico-dorsal sympathectomy:

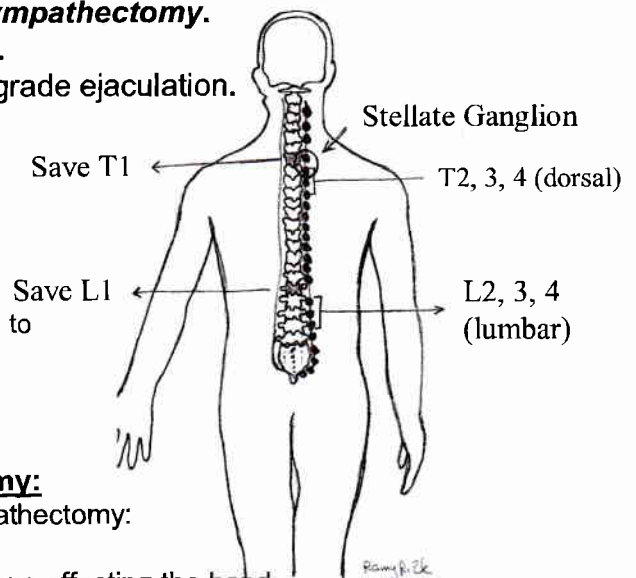
- a- Open cervico-dorsal sympathectomy:

○ Indications:

- Vasospastic conditions affecting the hand.
- Palmar (sometimes axillary) hyperhidrosis.

- b- Endoscopic transthoracic sympathectomy:

○ Indications: hyperhidrosis only.



▪ Lumbar sympathectomy:

- a- Open lumbar sympathectomy.
- b- Chemical sympathectomy: require the injection of small quantities of dilute aqueous phenol into the lumbar sympathetic chain under radiographic control.

3- level of amputation:

- a. If the popliteal pulse is felt, below knee amputation is done.
- b. If the popliteal pulse is not felt, above knee amputation is done.
- c. Forefoot amputation.



⇒ **The word "claudication" comes from**

- 1- The Latin "claudicare" meaning to limp.
- 2- The roman emperor Claudius walked with a limp, which was possibly due to poliomyelitis.

⇒ **DD of claudicating pain**

- 1. **Osteoarthritis:** pain on the first step
- 2. **Lumbar prolapse:** pain not relieved by standing still.
- 3. **Venous claudication:** ↑ on prolonged standing & ↓ on lying flat.
- 4. **Flat foot:** pain on standing or walking (due to pressure on plantar nerves).

⇒ **Factors affecting claudication distance:**

- 1- Speed of walk (high speed decreases it).
- 2- The presence of hills or soft floor (reduces it).
- 3- Upstairs movement.
- 4- Degree of ischemia.
- 5- General health such as anemia or heart failure.

⇒ **Arm claudication:**

- It may occur in upper limb in subclavian, axillary, brachial artery obstruction. The pain being brought on by such activities e.g. writing or manual labour.

⇒ **There is also venous claudication, resulting from inadequate venous drainage.**

- Warmth stimulates tissue metabolism → ↑ ischemia.
- Patient tends to uncover his limb (↓ metabolism) & put it in dependant position (↑ blood supply). – Rubbing of dorsum of foot

⇒ Subclavian steal phenomenon

- Thrombosis on top of atherosclerosis affecting the 1st part of subclavian artery before origin of vertebral artery.
- So the vertebral artery provide a collateral circulation by reversing its direction of flow.
- This may cause periods of cerebral ischemia due to shift of blood from the basilar artery.
- However, the classic syndrome of syncopal attacks and visual disturbance associated with arm exercise and diminished BP in affected limb is rare, asymptomatic reversal of flow is much more common.
- In symptomatic patient, PTA or operation is indicated

⇒ The acutely ischemic limb is frequently paralyzed and without sensation but chronic ischemia does not cause paralysis, but hyperesthesia is common especially in those areas of skin on borderline of gangrene.

⇒ Factors affecting the severity:

1. Size of the vessel occluded.
2. The collaterals available for blood flow.

⇒ Signs of impending limb gangrene:

1. Rest pain.
2. Edema.
3. Color changes.
4. Hyperesthesia.
5. Ankle pressure < 30 mmHg.
6. With or without ulceration.

⇒ Degree of ischemia can be determined by:

1. Degree of pain (claudication distance). Claudication distance < 50m and rest pain → severe ischemia.
2. Color changes: fixed color changes → severe ischemia.
3. Trophic changes.
4. Capillary circulation (sluggish > 2sec).
5. Venous filling: prolonged > 2min → severe ischemia.

⇒ The level of ischemia can be determined by:

1. Site of claudication.
2. Color changes.
3. Trophic changes.
4. Temperature changes.
5. Level of pulse.

Site of disease	Clinical finding	Type of operation
Aorto-iliac obstruction	- Claudication in both buttocks, thighs and calves (1st muscle to be affected is vastus medialis). - Femoral and distal pulses are absent in both limbs. - Bruit over aorto-iliac region. - Impotence (common in Leriche).	Technical support is not available <pre> graph TD A[Technical support is not available] --> B[Patient can withstand surgery] A --> C[Patient can not withstand surgery but with severe ischemia] B --> D[Aortofemoral bypass (Dacron bifurcation bypass grafting is the best for Leriche syndrome)] C --> E[Only 1 iliac artery is involved] C --> F[Both iliac arteries are involved] E --> G[Femero-femoral or ilio-femoral cross over bypass] F --> H[Axillo-femoral bypass] </pre> Technical support is available → PTA
Iliac obstruction	- Unilateral claudication in the thigh and the calf and sometimes the buttock. - Bruit over the iliac region. - Unilateral absence of femoral and distal pulse.	
Femoro-popliteal obstruction	- Unilateral claudication in the calf. - Femoral pulse is palpable with absent unilateral distal pulse.	Superficial femoral occlusion <pre> graph TD A[Superficial femoral occlusion] --> B[Mild] A --> C[Moderate or severe] B --> D[Conservative treatment] C --> E[Technical support] E --> F[Available] E --> G[Not available] F --> H[PTA] G --> I[Femero-popliteal bypass] </pre> Profunda femoris origin stenosis → small vein prosthetic patch angioplasty
Distal obstruction	- Femoral and popliteal pulses are palpable. - Ankle pulse is absent. claudication in calf and foot.	Bypass to a tibial vessel, even down to ankle level.

Diabetic Foot infection and gangrene

Definition

- Term used to describe complex pathology in a diabetic patient's foot. It is related to duration and control of disease.

- ▶ Ulcer
- ▶ Infection
- ▶ Vasculopathy
- ▶ Gangrene
- ▶ Mixed

Predisposing factors

➔ Diabetic patients are susceptible to serious foot infections due to:

- 1- Peripheral neuropathy: diminished sensations makes the patients unaware of foot injuries.
- 2- Vascular affection: due to premature atherosclerosis and microangiopathy.
- 3- Compromise of the immune system: both the humoral and cellular immunity are disturbed.

Presentation

- Neuropathic ulcer.
- Diabetic foot infection.
- Diabetic vasculopathy.
- Mixed types.
- Gangrene.

○ Neuropathic Ulcer

A. Causes:

- Patient is unaware of his foot → foot injury → neglected → ulcer

B. C/P:

- Painless, deep ulcer, may reach the bone, at pressure sites e.g heel, ball of the big toe.

C. Treatment :

- Control diabetes, Vit. B6, care of foot.



Ulcer in Diabetic Foot

N.B.

- 45% of diabetic foot ulceration are purely neuropathic in origin, 10% are purely ischemic, 45% are of mixed neuroischemic origin.

○ Diabetic Foot Infection (usually start by minor trauma or infection)

A. Causes:

- Decrease resistance (both humoral and cellular immunity are disturbed).
- Glycosylation of tissue.
- +/- ischemia.
- Neuropathy.

B. C/P:

- Persistent ulcer
- Severely swollen limb, red, hot & tender.
- Pus loculus, tissues becomes dark and slough.
- May spread → osteomyelitis, septic shock.

C. Treatment:

Prophylactic:

- Care of patient: control diabetes.
- Careful trimming of toes to avoid their injury.
- Avoidance of tight foot wear
- Early treatment of tenia pedis infection.
- Daily inspection of the feet to look for wounds and interdigital infections.
- Avoidance of walking barefooted.
- Daily feet care by washing, drying and powdering them.

Curative:

- Hospitalization.
- Rest in bed and elevation of the foot.
- Blood sugar is estimated and controlled better by crystalline insulin.
- Broad-spectrum antibiotics are started immediately before C&S. Anaerobes are commonly involved.
- **Drainage of infection and debridement:**
 - 1- Under general anaesthesia all pockets of pus should be drained. All sloughs should be excised. A gangrenous toe needs to be amputated leaving the wound open.
 - 2- If the patient is in septicemia, a major amputation is necessary to save the patient life.
 - 3- Repeated dressing and repeated debridement.
 - 4- When the wound becomes completely free of infection, plastic skin covering is considered to shorten recovery time.

○ Diabetic Vasculopathy

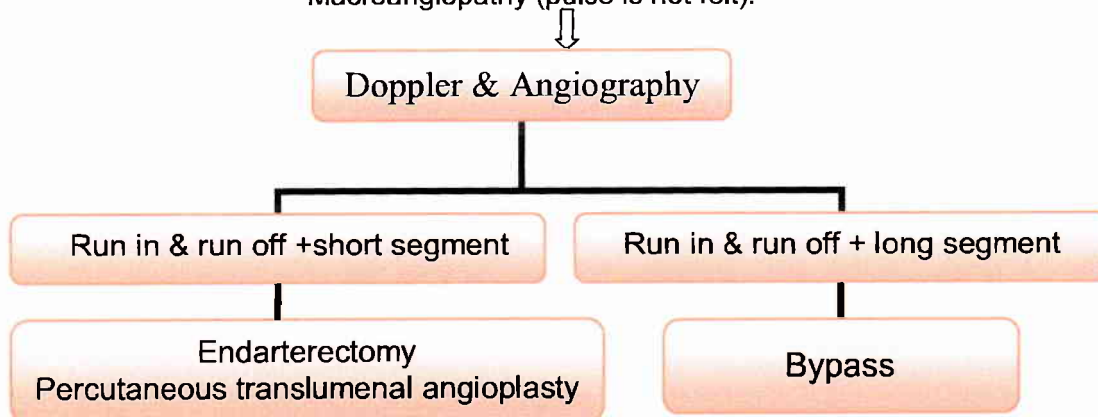
A. Cause

- Macroangiopathy (acceleration of atherosclerosis).
- Microangiopathy due to deposition of glycogen and lipids on the walls blood vessels.

B. C/P: of chronic ischemia (see before).

C. Treatment:

- Microangiopathy: (all pulses are felt, no bleeding from cut wound)
→surgical debridment, antibiotics, skin flap to promote healing .
- Macroangiopathy (pulse is not felt).



○ Mixed Type

- There is severe infection in addition to major vascular and neuropathic affection

○ Gangrene

- Wet gangrene: on top of infection.
- Dry gangrene on top of chronic ischemia.
- (C/P & TTT see gangrene).

Vasospastic Disorders

Definition

1. **Recurrent Ischemic attacks.**
2. **Precipitated by** exposure to cold or emotional stress.
3. **Common in UL & rare in LL.**
4. **It consists of:**

- 1 Pallor (white) → due to VC.
- 2 Cyanosis (blue) → due to sluggish blood flow.
- 3 Rubor (red) → due to reflex VD (accumulation of metabolites).

- ▶ UL > LL
- ▶ 3 colors (white, blue, red)
- ▶ Raynaud's: 1ry, 2ry
- ▶ TTT: Conservative, Surgery, Cause

DD

	Raynaud's Disease (1ry)	Raynaud's phenomenon(2ry)
Age & sex	young females (20 - 40 yrs)	Vary according to underlying cause
Cause	- Not exactly know, certain factors are suggested e.g.: 1- Increase sympathetic tone. 2- Psychological instability. 3- Abnormal sensitivity of small arteries and arterioles of hand to cold.	- Collagen → SLE, scleroderma. - Arterial obst. → Atherosclerosis, Buerger's disease. - Nerve injury → Thoracic outlet \$, carpal tunnel \$. - Environmental → Vibrating tools - Drugs → beta-blockers.
Distribution	- It affects UL (may affect LL). - It is almost always bilateral. - It is almost always symmetrical.	- May affect LL. - May be unilateral. - If bilateral, it is asymmetrical.
Trophic changes & gangrene	Absent or minimal.	More prominent.
Radial & ulnar pulse	Present.	May be absent.
Follow up	No progression & no evidence of underlying disease.	- Progressive. - manifestations of underlying dis.
Investigation	Should be directed to the suspected underlying cause	
Treatment	<u>Conservative (in early stages)</u> <u>Care of patient</u> - Stop smoking. - Avoid cold weather. - TTT of anemia if present. <u>Control diabetes</u> <u>Care of Hand</u> - Good hygiene, dryness of the hand and wear woolen gloves in winter - Hand exercise. <u>Drugs:</u> as chronic Ischemia + - Vasodilators → phenoxybenzamine, nifedipine. - Baby aspirin (prophylactic for fear of vaso-obliteration). - CCB. <u>Surgery (in severe cases)</u> - Cervicodorsal Sympathectomy. - Immediate results are good, but may recur after one or two years (due to denervation hypersensitivity).	- Treatment of the cause. - Vasodilators and beta-blockers may be used. - No benefit from sympathectomy as there may be: * Vasculitis. * Cryo-antibodies → cause VC in case of lowered blood temp. to 36-36.5° c

Acrocyanosis

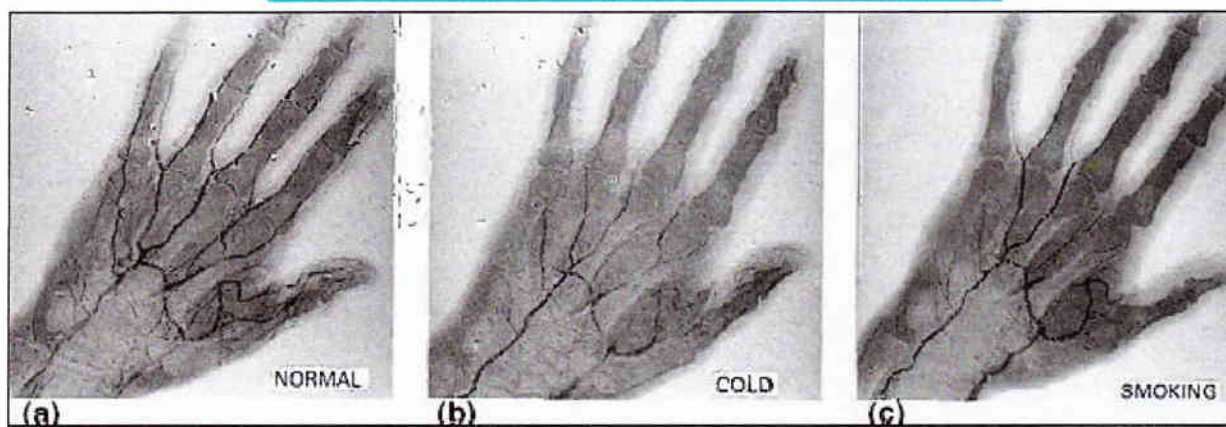
- It is due to hypersensitivity to cold. It differs from Raynaud's disease in:
 - Painless.
 - Not paroxysmal.
 - Cyanosis is marked and reticular.
 - Affects upper and lower limbs.
 - Associated with lower limb paraesthesia.
- TTT:** in severe cases → sympathectomy.

Frost Bite:

- C/O:** occurs due to cold exposure
- Pathology:** there is damage in vessel wall followed by transudation and edema
- C/P:** severe burning pain in affected part, blistering and gangrene may follow
- Treatment:** The proper emergency treatment is immersion of the affected part in warm water at 40 → 44 C



1ry Raynaud's in 24-year-old female



The induction of digital vasospasm by cold and smoking

- Apparently normal angiogram of 24 year old female with primary Raynaud's syndrome
- Intensive vasospasm prevents filling of digital vessels after cold exposure in the same patient
- cigarette smoking has the same vasospastic effect

Arteritis

Buerger's Disease

Etiology

➤ The etiology is unknown.

- Affects males only.
- Starts between 20-40 years old.
- Occurs exclusively in smokers; it may represent allergic reaction for nicotine.
- Interdigital fungal infection is a common association.

- ▶ ♂, 20 - 40 yrs
- ▶ Heavy smoker
- ▶ More distal
- ▶ Path.: Inflammation of NVB
- ▶ Arteriography: Corkscrew
- ▶ TTT: Sympathectomy, Amputation

Pathology

➤ See chronic ischemia.

Clinical Features

- The disease should be suspected in a young male who is a heavy smoker complaining of chronic ischemia in one or more limbs.
- The disease is preceded by migrating thrombophlebitis.
- Raynaud's phenomenon may be superimposed.

Differential diagnosis

	ATHEROSCLEROSIS	BUERGER'S DISEASE
Age of onset	Elderly	20-40 years
Sex	More in males	More in males
Etiology	HTN, DM,	Excessive smoking
Level of lesion	Aorto-iliac, femoro-popliteal or distal	Distal vessels with patchy distribution
Pathology	Mainly intimal (atheroma)	Inflamed neurovascular bundle and thrombi that block the lumen
Migrating thrombophlebitis	Absent	Usually present
Rest pain	May be present but late	Marked early feature

Investigations

➡ **ARTERIOGRAPHY**

- Is rarely needed to confirm the diagnosis.
- This is because the disease affects the distal vessels and the distal run-off is known to be absent.
- So there is no possibility for reconstruction, and consequently no need for arteriography.

Treatment

- 1- Smoking must be stopped.
- 2- Sympathectomy gives good results.
- 3- Amputation of one or more toes or digits is indicated for persisting pain or gangrene.

End Arteritis Obliterans of Syphilis

Arteritis of Collagen Disease

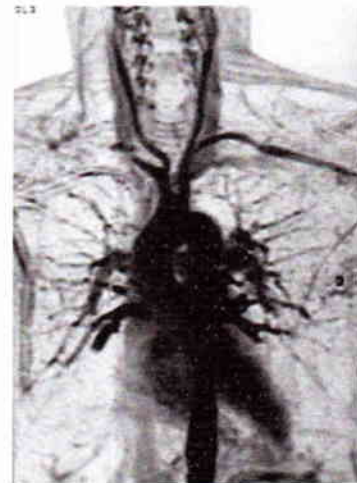
- Polyarteritis nodosa.
- Scleroderma.
- Rheumatoid arthritis and systemic lupus.

Infective Arteritis

- Bacterial invasion of vessel wall.
- Purpura fulminans.

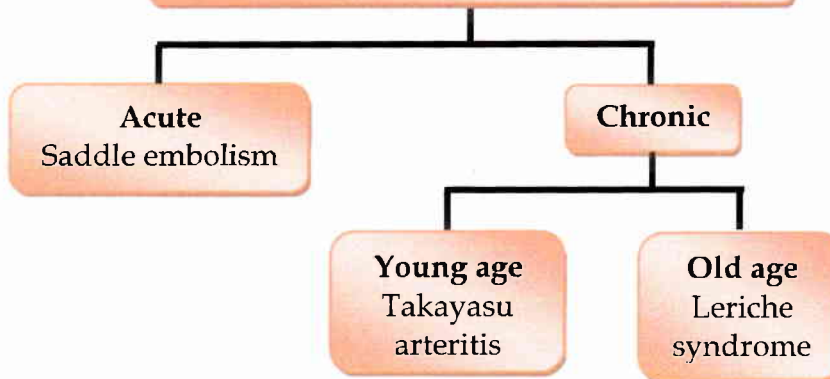
Takayasau's Arteritis

- (Pulseless disease of **young females**) autoimmune arteritis affecting the thoracic and abdominal aorta and their major branches and is usually fatal.



Takayasau's arteritis

Scheme for aortoiliac obstruction



Thoracic Outlet Syndrome

Introduction

The brachial plexus and the subclavian artery pass to the upper limb through a narrow triangle in the base of the neck. This triangle is made of **scaleneus anterior muscle anteriorly, scalenus medius muscle posteriorly and the first rib inferiorly**.

At this narrow space, compression of the nerve and artery may occur, causing symptoms and the development of the thoracic outlet syndrome.

Etiology

1. Cervical rib:

- It is an extra-rib → articulates with C7.
- Present in 0.5 % of population.
- May be bilateral. - Usually asymptomatic.
- The rib may be:
 - Complete → articulates anterior with manubrium or.
 - Incomplete → with a free bulbous or Fibrous band anteriorly.

- ▶ **Etiology:** Bone, Ms, N., A., Flesh
- ▶ **C/P:** Neuro, Art., Vein
- ▶ **Invest.:**
 - Bone (X-ray)
 - Nerve (N.C.V.)
 - Artery (arteriography)
- ▶ **TTT:** Conservative, Surgery (cause)

2. Scalene syndrome:

- Very strong scalene muscles.
- Weak shoulder girdle muscles.

3. Malunion of fracture clavicle.**4. Pancoast tumor.****5. Tumors or infection of the 1st rib.****6. Post fixation of the brachial plexus:**

- In this case the lower root of brachial plexus arises from T2 instead of T1, so becomes severely bended over the 1st rib.

Clinical Picture

- The condition occurs more frequently in females.
- It is usually unilateral affecting the right arm (dominant arm), but is sometimes bilateral.

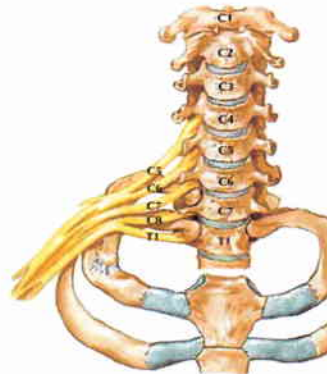
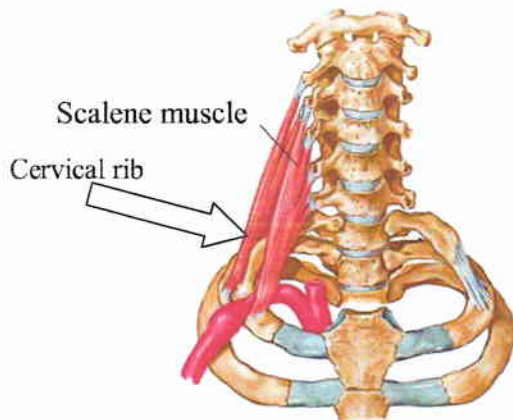
	It is usually unilateral affecting the right arm (dominant arm), but is sometimes bilateral.		
	Neural	Arterial (less common)	Venous (very rare)
Due to	Compression of the lower trunk of the brachial plexus (C8 & T1). It is the main structure to be compressed.	Compression of subclavian or axillary artery.	Compression of the subclavian or axillary vein.
Symptoms	1- Sensory → tingling & numbness along the ulnar side of hand & forearm. 2- Motor → Weakness & atrophy of the small muscles of the hand.	1- Chronic ischemia of upper limb: a- Claudication pain with exercise. b- 2ry Raynaud's phenomenon (on exposure to cold) (due to irritation of sympathetic fibers supplying U.L.) 2- Post-stenotic dilatation: may develop a thrombus inside. This thrombus may send a shower of emboli to the index and middle finger as they are direct continuation of the brachial artery. This emboli can produce digital gangrene.	1- Subclavian or axillary vein thrombosis (Effort thrombosis). C/O: acute pain and swelling (DVT). 2- Chronic venous insufficiency. C/O: chronic heaviness and swelling.
Signs	- ↓ Sensation. - ms weakness & atrophy.	- Unequal pulse on both sides. - Positive Addison's Test → ask the patient to: - Extend the head. - Look at opposite side. - Take a deep breath. - Then palpate radial pulse while arm is pulled down. If positive → radial pulse becomes weak	
	- A palpable cervical rib may be felt at root of neck especially if bulbous.		

DD

- Other causes of localized pressure on nerve: cervical spondylosis, carpal tunnel syndrome.
- Other causes of Raynaud's phenomena: Raynaud's disease, systemic lupus.

Investigations

1. **Plain x-ray** → cervical rib.
2. **Arteriography** → show post - stenotic dilatation.
3. **Nerve conduction studies** →
Reveal prolonged nerve conduction time.
→ **Very useful to differentiate carpal tunnel syndrome from thoracic outlet syndrome.**

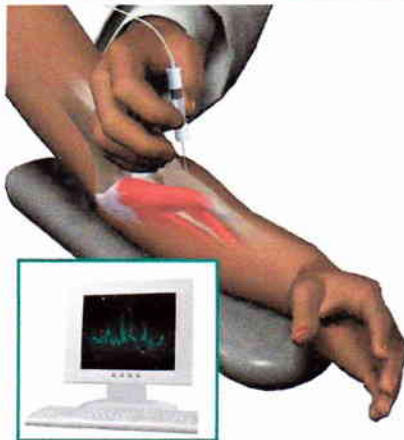


Cervical rib compresses subclavian artery & there is poststenotic dilatation

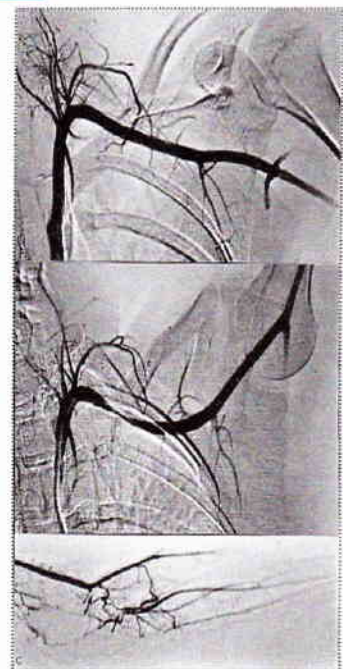
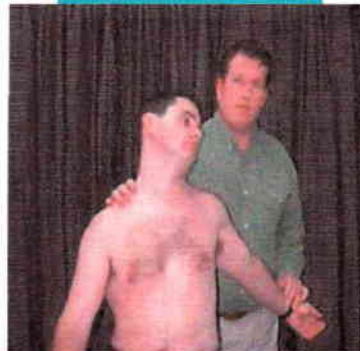
Compression of the lower trunk of the brachial plexus

Strong Scalene muscle

Electrical Studies → reveal prolonged nerve conduction time



Addison's



Treatment

Mild cases with neurological symptoms

Physiotherapy & shoulder exercises → to develop shoulder girdle muscles.

Severe cases with vascular symptoms or failure of conservation

Surgical Treatment → results are bad with many complications.

- Resection of the cervical rib (with its periosteum).
- Excision of the 1st rib to relieve compression (transaxillary)
- Division of scalenus anterior muscle (scaleneotomy).
- Osteotomy and internal fixation of clavicle.
- Radio and chemotherapy for pancost tumor.

Carotid Body Tumor (Chemodectoma)

Definition

- Rare slowly growing tumor arising from the chemoreceptors present at the bifurcation of carotid artery

Incidence

- Males = Females
- Usually at 20 years of age
- 5% malignant (percentage ↑ with size)

Clinical Picture

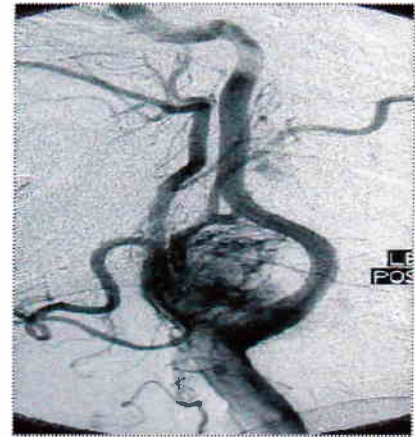
- Middle age female or male with slowly growing swelling adjacent to hyoid bone, anterior to sternomastoid muscle, smooth, compressible, pulsatile, moves from side to side not vertical

Investigations

- CT scan
- Angiography: widening the carotid artery bifurcation

Treatment

- Excision with preservation of ICA



Carotid body tumor

Gangrene

Definition

- Macroscopic death of tissues due to loss of blood supply and is usually associated with bacterial invasion and putrefaction, necrosis may be used synonymously.

► **Etiology:** A., V., N., Trauma, Infection

► **Classification:** • Moist

- Dry
- Others

► **TTT:** Conservative, Non-conservative, amputation

Site

- It often affects the distal part of a limb because of arterial obstruction (thrombosis, embolism or arteritis)

Etiology

1. **Ischemic** → thrombosis, embolism, vasospastic disease.
2. **Neuropathic** → syringomyelia, leprosy.
3. **Venous Gangrene** (see below).
4. **Traumatic** → direct: e. g. bed sores,
→ indirect: e. g. arterial injuries.
5. **Infective** → Specific infection: e.g. clostridial gas gangrene.
→ Non-specific infection e.g. carbuncle, anaerobic cellulites.
6. **Physiochemical** → burns, caustics, frostbite,...

Types

(There are 3 types of gangrene depending on rate of local death in the presence of infection)

	1- Dry Gangrene	2- Moist Aseptic Gangrene
Pathogenesis (cause)	Chronic ischemia → allows dryness of tissues.	- Acute ischemia - Chronic ischemia with pre-existing edema (cardiac, DVT)
Pathology		
Putrefaction	Minimal.	Marked
Odor	little or no odor	Very offensive
Gross	- Dry. - Hard. - Dark in color. - Mummified. - Wrinkled.	- The part remains of the same size and consistency. - Colour: at 1 st dead white the purple or greenish black.
Line of demarcation	well defined	ill defined (no time for evaporation)
Fate	Separation → - Due to presence of line of demarcation → appears between the dead & viable tissues. - Leaving a conical stump (deeper tissue has better blood supply).	Spread → by - Direct extension. - Skipped lesions.
C/P	1. The five cardinal signs of local death are: Lost (p ulsation, S ensation, H eat, F unction of affected part) fixed color changes. Press & see How Colour fades NB. Patient suffering from threatened gangrene have all the above signs but the tissues are still viable and local pressure causes some modification of color, which returns when the pressure is released. 2. The affected part is → see gross.	
	3. Minimal toxemia → better general condition.	3. Severe toxemia → poor general condition.
Treatment	See chronic ischemia - Limb salvage (conservative amputation). - Non-conservative amputation.	- Amputation till the level of pulsation (acute condition) (non-conservative amputation). - Later on ttt of the cause if possible.

3- Moist Septic Gangrene:

- **Etiology:**
 - Infection of sterile gangrene (2^{ry} infective gangrene).
 - Infection of tissues with virulent organisms leading to gangrene (1^{ry} infective gangrene)
- **Clinical Picture:**
 - The affected limb becomes swollen, edematous and markedly inflamed.
 - The skin appears moist with bullae filled with serum.
 - The limb emits a very offensive odour and may crepitate due to gas formation.

- **Line of demarcation** : between dead and healthy tissue
- **Line of separation**: level at which the necrotic tissue separates from healthy tissue.

4-Special Types of Gangrene:

a- Decubitus Ulcer (Bedsore):

Site

- Occurs over bony prominence (sacrum, ischial tuberosity or heels)

Etiology

- Due to prolonged pressure.
- Following immobilization of paraplegic patients, elderly & diabetics.

Treatment

1. Prevention.

- Air mattress.
- Skin should be kept dry & clean.
- Frequent change of position every 2 hours.

2. Curative

- Debridement .
- Leave the wound open until healing + repeated dressing with glycerin magnesia to help separation of the slough.
- Then, antibiotics (can not be given alone as it is dead tissue so it will not benefit from it)
- Skin flap.



b- Idiopathic Gangrene of the Scrotum

(Fournier's Gangrene):

- Sudden onset of scrotal inflammation + sudden onset of gangrene
- Due to mixed infection (synergism) by Strept, Staph., E. coli, Cl. Welchii,....etc.
- May be associated with necrotizing fasciitis

Treatment.

- Antibiotics & wide surgical excision.
- Later on, skin graft may be needed to cover the testis.



c- Venous gangrene (rare condition)

- 1- Caused by extensive thrombosis of the major peripheral veins (phlegmasia cerulea dolens)
- 2- **Treatment:**
 - The limb is elevated and anticoagulant therapy is started.
 - Thrombectomy or fibrinolytic treatment should be considered.

Aneurysms

Definition

- **Pathological definition:**
A sac filled with blood communicating with an artery.
- **Practical definition:**
Permanent localized dilatation of an artery 1.5 time than normal

- ▶ **Types:**
 - Etiology: cong., path., traumatic
 - Wall: false, true
 - Shape: saccular, fusiform, dissecting
- ▶ **C/P:** Asymptomatic, Swelling, Ischemia
- ▶ **Comp.:** Rupture, Pressure, Ischemia, Infection
- ▶ **Invest.:** U/S (if suspected), CT (accurate)
- ▶ **TTT:** Conservative, Surgery

Types

I - According to Etiology:

1. Congenital.

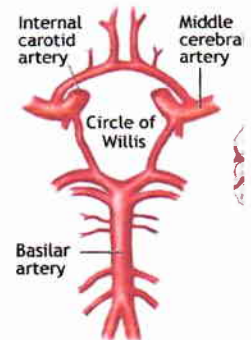
e.g. aneurysms in circle of Willis "Berry's aneurysm" rupture causing spontaneous subarachnoid hemorrhage.

2. Pathological due to:

- Atherosclerosis (commonest cause).
- Trauma.
- Syphilis.
- Mycotic (i.e. arterial wall is weakened by infection e.g. following embolization from SBE.
- Collagen disease e.g Ehlar Danlos, Behchet.

3. Traumatic:

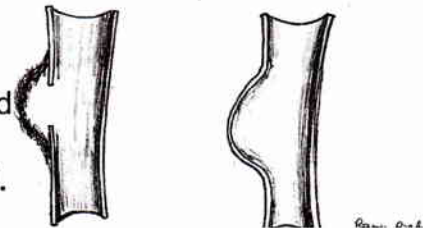
- A blunt trauma may weaken part of its wall . Later this weak area progressively yields leading to aneurysmal dilatation.
- A penetrating injury to the artery leading to hematoma surrounding the artery .Later on, this clot is surrounded by organized fibrous capsule leading to false aneurysm.



II - According to the Wall of Aneurysm:

1. **False:** organized hematoma with fibrosed wall and compressed tissue around it.

2. **True:** wall consists of all layers of the arterial wall.



III - According to Shape:

1. Saccular.

2. Fusiform.

3. Dissecting.

False Aneurysm

True Aneurysm

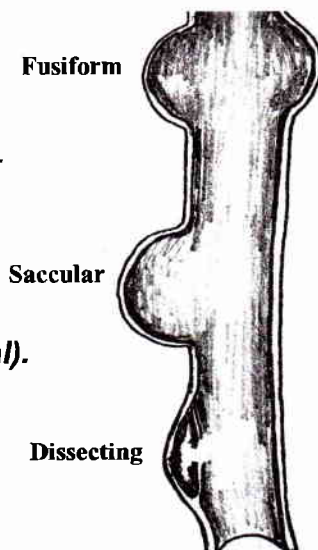
Clinical Picture

Type of patient

- **Age:**
 - old age: atherosclerosis and syphilitic aneurysm.
 - Yong age: traumatic aneurysms are common.
- **Sex:** more common in males.

Symptoms

- **May be asymptomatic.**
- **Swelling (external) or pressure manifestation (internal).**
- **Ischemic manifestations** due to:
 - The cause (atherosclerosis) → chronic ischemia.
 - The patient may be presented by complications:
 - Embolism → acute ischemia.
 - Rupture → acute ischemia, shock.



Signs

- **General:** tachycardia in case of large aneurysm, associated heart disease.

- **Local:**

Inspection

- Swelling.
- Along the course of an artery.
- Expansile pulsation (systolic) (most important sign)

Palpation

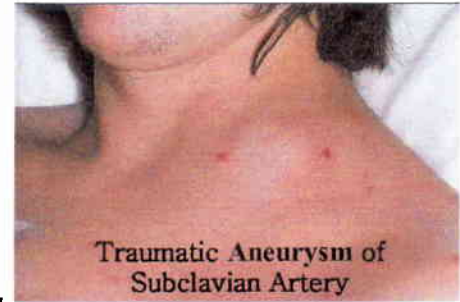
Consistency → cystic.

Movement → across but not along an artery.

Compression

- Totally compressible.
- Partially compressible → thrombosis.
- Proximal compression → on feeding artery → ↓ size & pulsation becomes weak.
- Distal compression → on affected artery → ↑ size & become tenser.

Auscultation → soft systolic bruit may be heard over it.



Traumatic Aneurysm of Subclavian Artery

DD

1. **Swelling over an artery** → gives transmitted pulsation; pressure on proximal artery does not change size.
2. **Vascular tumors** → heterogeneous consistency e.g. osteosarcoma.
3. **A-V fistula.**
4. **Abscess**
5. **Serpentine arteries esp. in common carotid artery.**

Complications

- Rupture (most serious complication) → fatal hemorrhage (suspect if diameter >6 cm, ↑ elastase in blood)
- Infection: may lead to rupture and 2ry hemorrhage.
- Pressure manifestations:
 - o Nerve → sensory, motor loss.
 - o Bone → erosion.
 - o Vein → obstruction, thrombosis.
- Ischemia due to:
 - o Thrombosis.
 - o Embolism.
 - o Associated atherosclerosis.

Investigation

1. **Plain x ray:** may accidentally discovered calcification in wall of artery.
2. **U/S** → best if aortic aneurysm is suspected → non-invasive, cheap & can determine the diameter and extension of the aneurysm.
3. **CT scan:** is the accurate investigation to determine the diameter of the aneurysm
 - CT accurately determines the true extension of the aneurysm
 - In aortic aneurysm It is important to determine if the upper limit of the aneurysm is below, at, or above the renal arteries because the operative treatment is different in each situation
 - It is important to determine if the iliac arteries are also involved.
4. **Angiography:** Angiography does NOT show the true diameter of the aneurysm. It only shows the diameter of the patent lumen
Some times fails to demonstrate the aneurysm due to clotted blood inside it.

- Recently, CT angiography with 3D reconstruction of the arterial tree is gradually replacing conventional CT and angiography
- It is more accurate than older CT & less invasive than angiography as the dye is injected intravenously

Treatment

1. Conservative.
2. Surgery:

Indications

1. Symptomatic aneurysm.
2. Asymptomatic if the diameter is >55mm.
3. Asymptomatic in high-risk patient e.g. hypertension.

Technique

- Excision → for small or unimportant artery.
- Excision & grafting → for aneurysm of large artery.
- Exclusion graft (open the sac, place the graft then close the sac)

Endovascular surgery: intraluminal self-inflatable graft.

Abdominal Aortic Aneurysm (AAA)

The commonest type of aneurysms

Definition

- See before + affecting abdominal aorta.

Causes

- Mainly due to atherosclerosis. (95%)
- Rarely due to other causes (mycotic, syphilitic, collagen disease).

Pathology

- Site: 95% below origin of renal arteries.
- Types: see before.

Clinical Picture

- **Silent in 75 % of cases.**
- If symptomatic → pain (commonest symptom) → vague abdominal pain with flank and backache (due to vertebral compression).
- May present with complications →

○ Rupture :

Types of rupture aneurysms:

- 1- **Intra-peritoneal rupture** (free rupture) is fatal. Patients usually don't reach hospital

2- **Retro-peritoneal rupture** is an extreme emergency. It has a high operative mortality (this the reason way we prefer to repair large (>4cm) asymptomatic aortic aneurysms as set operations have much lower mortality.

Clinical Picture of ruptured AAA

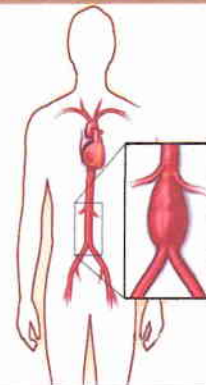
Symptoms

- Sudden severe onset of epigastric pain.
- Collapse with severe hypotension.
- May have history of aneurysm under surveillance.

Signs

- Cardinal sign: unexplained rapid onset hypertension, pain and sweating.
- A pulsatile abdominal mass is not always present.

- ▶ **Etiology:** 95% atherosclerosis
- ▶ **Pathology:** 95% below renal arteries
- ▶ **C/P:** 75% silent, pain, swelling
- ▶ **Invest.:** CT scan, U/S
- ▶ **TTT:** Conservative, Surgery



Abdominal Aortic Aneurysm



Investigations

- CT scan is the investigation of choice to investigate painful, leaking or ruptured aneurysm
 - Arterio-aortic embolism: **mural thrombus** within the aneurysm can detach and cause distal emboli
- Embolization may be spontaneously (blue toe syndrome) or iatrogenic during surgery (trash foot).

DD

- Pseudopancreatic cyst: transmitted pulsations.

Complications

- See before

Investigations

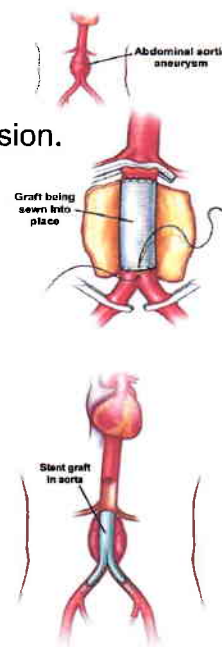
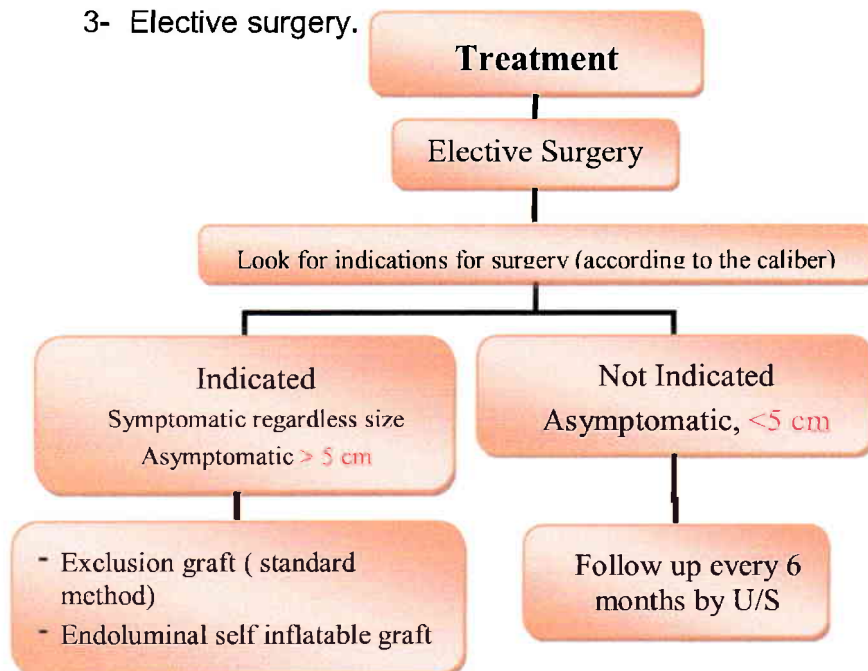
- For aneurysm → U/S, CT, MRA, Angiography.
- For other system affection → ECG, CBC, lipid profile.

- US is the screening test of choice to documents or to rule out the presence of aneurysm
- Arteiography may appear normal despite presence of large AAA if the aneurysm is occupied by thrombous so it is not used to diagnose AAA or to assess its dimension, is only of value if the patient is suspected to have associated occlusive disease.

Treatment

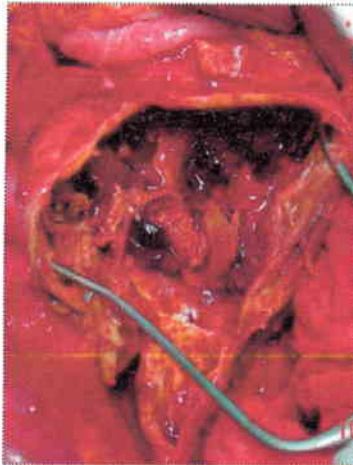
► Plain of management:

- 1- immediate surgery for patient with diagnosis of rupture.
- 2- Urgent surgery for patient with symptoms of acute expansion.
- 3- Elective surgery.

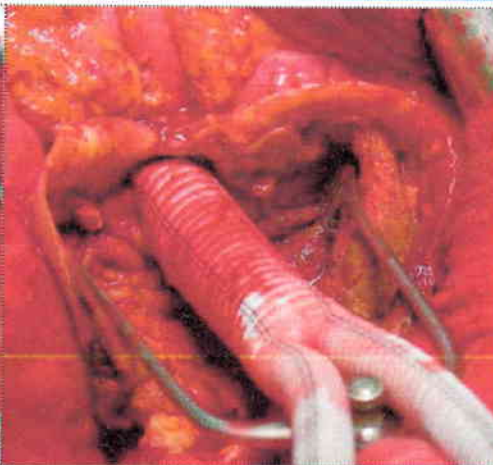


N.B. Endovascular repair of AAA via insertion of endoluminal stented graft through bilateral femoral arteriotomies is expensive and recurrence of aneurysm can occur so, only indicated for:

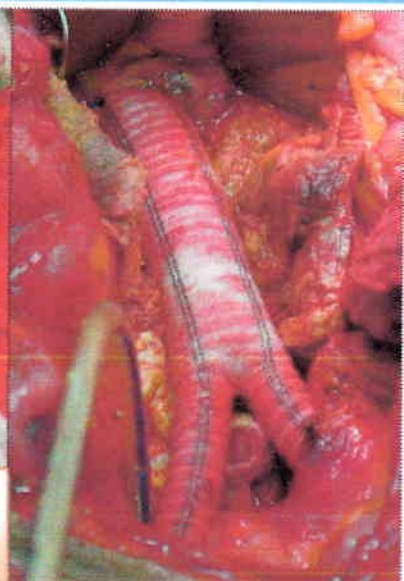
- 1- High risk patient who can not tolerate anaesthesia or open surgery
- 2- High risk local abdominal factors (intra-abdominal infection)



Aneurysm opened
Note the clots and cholesterol debris. These may cause distal embolization



The bifurcated graft is first sutured to the aorta **from inside** of the aneurysm



In this patient, the 2 limbs of the graft are then sutured to both iliac arteries

Arterio-Venous Fistula

Definition

- It is an abnormal shunt between an artery & vein, it may be congenital or acquired.

A- Congenital A-V fistula

- They are usually multiple connections, affecting small vessels (does not cause ischemia) and may give rise to local gigantism.
- Usually clinically undetectable but may be manifested by varicose vein.
- Often associated with port wine hemangioma.
- Treatment:**
 - Treatment by surgery is generally useless, as ligation of one connection will cause other connection to open up, therefore the affected part should be protected from trauma.

B- Acquired A-V fistula (traumatic)

- They are usually single, affecting large or small vessels (causes ischemia); it is common in the thigh (butcher's thigh), axilla or neck.
- Pathological types:**
 - Arteriovenous aneurysm: the communication between the artery and vein is not direct and there is an intervening hematoma, which later organizes.
 - Aneurysmal varix: the communication between the artery and vein is direct and there is no swelling in between.

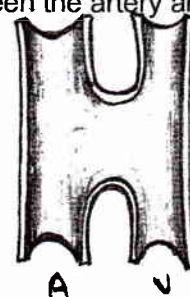
3. Clinical Picture:

⇒ **General** → tachycardia, ↑ cardiac output with high systolic blood pressure and low diastolic blood pressure → water hammer pulse.

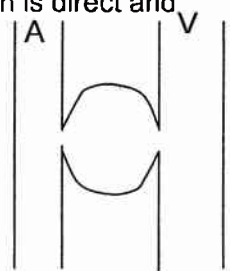
⇒ **Local**

Inspection

- Pulsating swelling.
- Distally → Varicose veins & ischemia.



Aneurysmal varix



A-V aneurysm

Palpation

On the swelling → thrill.

Compression

- Totally compressible.
- Partially compressible → thrombosis.
- On feeding artery (at site of fistula) → ↓pulse "Branham's bradycardia" (AV fistula → ↑COP, on compression → ↓COP → ↓pulse).

Auscultation → machinery murmur may be heard over it.

⇒ **Distal**: secondary varicose veins, ischemia.

4. Investigations

- 1- **Arteriography** → shunting of the blood from the artery to the vein or show both arteries and veins at the same time due to rapid venous filling.
- 2- **Doppler** → can localize the site of the fistula.

5. Treatment

1. **Reconstruction of the artery & vein.**
2. **Quadruple ligation:**
 - If Reconstruction is difficult (as in HF).
 - Unimportant artery.

Congenital A-V fistula of the hand. The thumb, index fingers, and thenar eminence are enlarged, veins are distended, and the thenar eminence is pulsatile



Endovascular Surgery

Definition

- Management of vascular diseases percutaneously through a puncture to deal with a lesion in a remote site.

Classification

- System: 1- Arterial.
2- Venous.
- Purpose: 1- Diagnostic.
2- Therapeutic.

Therapeutic procedures

- 1- Angioplasty.
- 2- Intravascular stents.
- 3- Thrombolytic therapy.
- 4- Atherectomy.
- 5- Peripheral laser angioplasty.
- 6- Endoluminal grafts.
- 7- IVC filters.
- 8- Catheter vascular access implantation of a catheter to access a central vessel for either:
 - a- hemodialysis in chronic renal failure.
 - b- Chemotherapy.

BALLOON ANGIOPLASTY (PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY= PTA)

➡ Indications:

- 1- Critical ischemia.
- 2- Severe claudication that interferes with patient's work or style.

➡ Ideal lesion:

- 1- At least 5mm beyond origin of an artery.
- 2- Stenosis <5cm length.
- 3- Best in iliac artery.

INTRAVASCULAR STENT

➡ Aim:

- May inserted in the same session after PTA to prevent its drawbacks (dissection and elastic recoil) in order to maintain its patency of the artery.

➡ Types:

- 1- Balloon-expandable stents.
- 2- Spring-loaded, self-expanding stents.
- 3- Thermal memory stents.

INFERIOR VENA CAVAL FILTERS

➡ Indications:

- 1- DVT or documented pulmonary embolism in a patient with C.I to anticoagulant therapy.
- 2- Recurrent pulmonary embolism.
- 3- Chronic pulmonary embolism in patients with pulmonary hypertension and cor-pulmonale.
- 4- Complications of anticoagulant therapy that forced to be discontinued.

ENDOLUMINAL GRAFTS

➡ Principle:

- A combination of a stent with prosthetic graft positioned with a delivery system.

➡ Uses:

- 1- Exclusion of A.A.A.
- 2- Support of the intima of an artery after balloon dilatation in some cases.

THROMBOLYTIC THERAPY

➡ Indications:

- 1- Acute DVT.
 - 2- Acute thrombotic ischemia.
 - 3- Acute coronary occlusion.
 - 4- Occlusion of vascular grafts.
 - 5- Pulmonary embolism.
 - 6- Acute embolic ischemia.
 - 7- Restoring patency of recently occluded central venous catheters.
- It is better done in an I.C.U under close observation.

➡ Contraindications:

- 1- Active bleeding.
- 2- Recent stroke.
- 3- Major operations within 2 weeks.
- 4- Infective endocarditis.

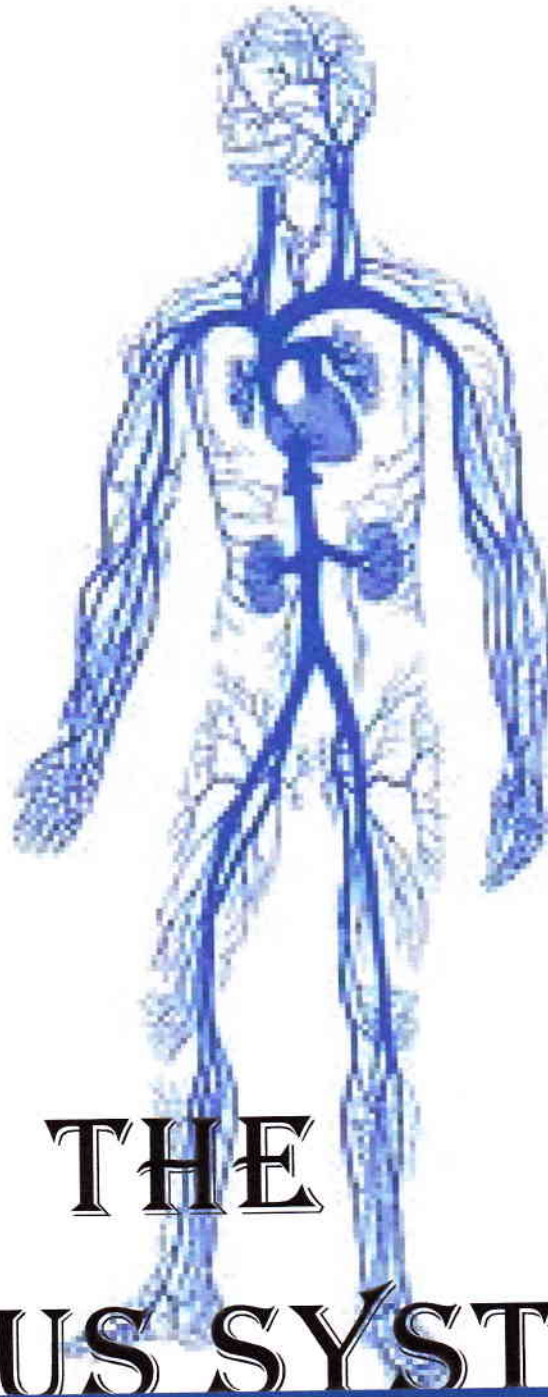
➡ Agents:

- Streptokinase, urokinase and recombinant tissue plasminogen activator.

Points for MCQ

- The commonest site of atherosclerotic occlusion in the lower limbs is distal superficial femoral artery.
- In diabetic patients with critical limb ischemia, the ankle/brachial index may be falsely high, so they may develop calcification of leg arteries.
- Approximately 50% of popliteal artery aneurysms are associated with aneurysms at other sites.
- The 1st thing to do after diagnosis of acute L.L. ischemia is to give the patient heparin to prevent clot propagation.
- Thrombolysis is contraindicated in patients with history of stroke within the previous 2 months or major surgery within the previous 10 days because of risk of bleeding.
- Aorto-iliac reconstruction surgery has good long-term outcome.
- Intra-arterial thrombolysis is best achieved with pulse-spray TPA.
- In fat embolism the fat is most likely to arise from chylomicrons.
- Therapeutic embolization is used for arrest of hemorrhage, TTT of A-V malformations & shrinkage of tumor growth.
- Venous gangrene occurs in presence of venous & arterial occlusion, embolism or diabetes.
- Bowel strangulation is an example of moist gangrene.
- Cessation of cigarette smoking is the 1st therapy for Buerger's disease.
- Buerger's disease is characterized by recurrent exacerbations & remissions.
- Intermittent claudication is usually the 1st symptom in Buerger's disease & it may end in gangrene.
- Endovascular Aortic Aneurysm Repair (EVAR) leads to absolute reduction in early mortality (within 30 days) compared to open repair

CHAPETR 2



THE VENOUS SYSTEM

General Principles of Venous Thrombosis

1- Sites:

- a- **Superficial veins.**
- b- **Deep veins:** occur mainly in the calf or iliofemoral veins. Less common sites are IVC, subclavian, axillary or portal veins.

2- Predisposing factors (Virchow's triad):

1. Velocity: slow circulation: Prolonged rest in bed.

- Postoperative: (Pelvic operations, hip replacement, visceral malignancy).
- Heart failure.
- Obesity.
- Pregnancy.

2. Viscosity: hypercoagulability:

- ↑ Blood elements : polycythemia , thrombocytosis, leukemia
- ↓ Plasma: burn.
- ↓ Fluids: dehydration.
- ↓ Natural anticoagulant : deficiency of protein C, S , antithrombin III (Recurrent DVT in young).
- Malignancy esp. ovarian malignancy, lupus anticoagulant.
- Drugs: contraceptive pills.

3. Vessel wall: injury of vascular endothelium:

- Inflammation.
- Trauma.
- Previous DVT → the **MOST** important cause.

3- Types:

- a- **Phlebothrombosis:** with this type, the venous thrombosis is not associated with infection. Therefore, the thrombus will not be adherent to the vein wall and it may detach resulting in pulmonary embolism.
- b- **Thrombophlebitis:** the venous thrombosis is associated with inflammation of the affected vein, so the thrombus will be adherent to the vein wall.

Superficial Thrombophlebitis

Predisposing Factors

- Vein → Varicose vein.
- Vein → damaged by intravenous cannula,
- Vein → near by a septic focus as an abscess.
- In association with Buerger's, polycythemia, PAN, visceral malignancy.

Clinical Picture

Symptoms

- **General** → FAHM.
- **Local** → pain & tenderness at the site of the affected vein.

Signs

- The vein is felt as tender, firm, cord like structure.
- The overlying skin is red & edematous.

DD

- Lymphangitis.

Complications

1. **Suppuration** & local abscess formation.
2. **Extension to deep veins** via communicating veins.
3. **Pulmonary embolism** (very rare because the thrombus is very adherent to the vessel wall except big veins as saphenous vein).

Treatment

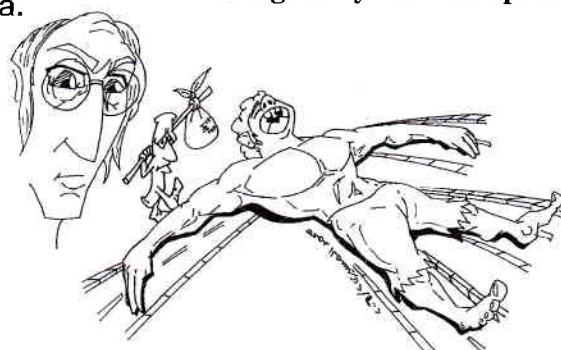
1. **Rest & compression by elastic stockings or compression bandage.**
 2. **Anti-inflammatory** drugs e.g. aspirin.
- 1 & 2 are usually enough for treatment of majority of cases. The following are given only under special conditions:
3. **Antibiotics** (only if there is evidence of infection).
 4. **Anticoagulant therapy** (heparin and warfarin) is given in severe cases (ascending thrombophlebitis or if associated DVT).
 5. **Surgery:** Prophylactic sapheno-femoral or sapheno-popliteal disconnection is done if there is tenderness less than 10cm from the junction.

Thrombophlebitis Migrans

- It is a type of superficial thrombophlebitis that resolves spontaneously in a few weeks to reappear in another area.
- **It occur with:**
 1. Buerger's disease.
 2. It is the earliest sign of visceral cancer (stomach, pancreas) "**Trousseau's sign**" due to break down of the tumor which causes hypercoagulation of the blood.
 3. Polycythemia & polyarteritis nodosa.
 4. Ulcerative colitis & SLE.

Migratory Thrombophlebitis

Superficial thrombophlebitis travels from vein to vein



Deep Venous Thrombosis

Incidence

- The true incidence of DVT is not exactly known as many cases pass unnoticed, however 10% of patients who die in hospital, the cause of death is PE following DVT.
- DVT is more common on the Lt. side because of the anatomical fact that the Rt. common iliac artery crosses over and compresses the Lt. common iliac vein

- ▶ 10%
- ▶ **Mainly asymptomatic**
- ▶ **C/P:** Δ Pain, Swelling, Tenderness
- ▶ **Comp.:** General (PE), Local
- ▶ **Invest.:** Duplex, Spiral CT
- ▶ **TTT:** prophylactic, curative
[Anticoagulants, Filter, Fogerty, Fibrinolytics]

Etiology

Virchow's triad (see before).

Pathology

- 1- The process usually starts in the calf venous sinuses or in the iliac and femoral veins by adherence of platelets to the endothelial surface forming grey cluster.
- 2- Fibrin and RBCs are deposited as layers in-between the platelets giving a laminated appearance known as the lines of Zahn.

- 3- When the vein is totally occluded, non adherent, jelly-like propagated thrombus spreads up the vessel as far as the next major tributary. This thrombus is dark red and consists **only of fibrin and red cells**. At this stage **the thrombus is loosely attached** and it can be easily detached leading to pulmonary embolism.
- 4- Later, the thrombus becomes tightly adherent to the vein wall by fibrin deposition. It then organizes and contracts thus producing destruction of the vein valves which is responsible for the development of **post-phlebotic limb**.
- 5- Later on the process of fibrinolysis and phagocytosis starts and helps in **recanalization** of the vein but the valves are permanently destroyed. 90% of occluded veins will be recanalized within 9 months while 10% of affected veins remain occluded for life.

Clinical Picture

1- Asymptomatic Group

- Most cases are silent.
- The first presentation may be PE or postphlebotic limb.
- Suspected by unexplained fever and tachycardia.

2- Classical Picture Group

(Usually manifested clinically on 3-7th day postoperatively)

> General:

- Mild unexplained fever.
- Tachycardia out of proportion to fever (lysis of thrombus → release of pyrogens & toxins).

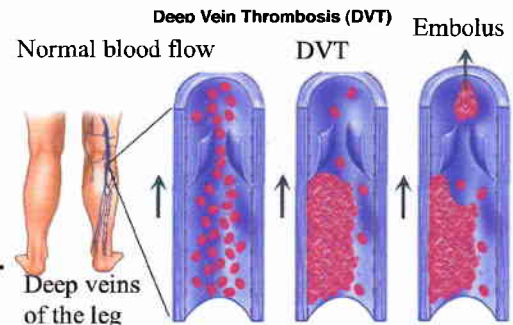
> Local:

- Classical triad of pain, swelling, tenderness.
- Depends upon the site of the thrombosis.
 - 1- **Pain:** there is usually aching discomfort and tightness in the involved calf or thigh, which are aggravated by muscular exercise.
 - 2- **Swelling** (it is the most reliable physical sign)
 - In calf thrombosis → swelling is limited to foot and ankle.
 - In femoral thrombosis → swelling involves calf and lower part of thigh.
 - In iliofemoral thrombosis → swelling affects the whole lower limb.
 - 3- **Tenderness:** present on grasping the affected calf or thigh.

NB e-thrombosis: blood clots occurring in sitting on computers for prolonged periods of time

Homan sign

- Pain is felt in calf on dorsiflexion of the foot; it is not a reliable sign as it may be falsely positive in cases of calf muscle contusion or cellulitis.



3- The complication group

a- General → pulmonary embolism.

Summary of Lymphoma

	Hodgkin's	Non-Hodgkin's
Macroscopically A) L.N. - Sites: - CC's: - Cut section: B) Spleen, Liver intestine & BM	- Usually lower deep cervical → axillary → mediastinal - Enlarged + satellites, discrete early → matted later. - Pink homogenous with loss of architecture.	- More than one group (multicentric). - Early discrete → amalgamated later.
Microscopically	Characteristic giant cell (Reed-Sternberg cell) with lymphocytes, histiocytes & PNL (pleomorphism).	
Histological types	1- Lymphocytic predominance 2- Nodular sclerosis 3- Mixed cellularity 4- Lymphocytic depletion	1-Lymphocytic lymphoma. 2- Histiocytic lymphoma. 3- Mixed cellularity. 4-Burkitt's lymphoma.
Clinically: 1-Age & sex: 2-Constitutional symptoms: 3-Swelling: 4-Pain: 5-Pressure manifest.	- Adolescent & middle age (bimodal) - ♂ - Fever → irregular. → Pel-Ebstien. - Itching (may be presenting \$). - Loss of wt. (> 10% in 6 m.) - Anemic manifestations. - Slowly progressive, rubbery → matted later. - Pain & itching (especially after alcohol intake "Gordon's test" or late after infiltration) - Mediastinal L.Ns → dyspnea, dysphagia, hoarseness & Horner's.	- Extreme of age - ♂ - Rapidly progressive , hard → Amalgamation later. Late after infiltration. - As Hodgkin's - As Hodgkin's but more common.

b- Local

a) Early:

1. **Phlegmasia alba dolens:** massive iliofemoral DVT may be associated with severe arterial spasm and lymphangitis, so the limb becomes pale, white and massively swollen with absent peripheral pulsation.
2. **Phlegmasia cerulea dolens:** massive iliofemoral DVT may be associated with severe congestion and cyanosis and the whole lower limb looks massively swollen and blue and if not probably treated, it may lead to venous gangrene.



b) Late:

1. **Post phlebotic limb** (secondary V.V due to destroyed valves 2ry to recanalization)
2. **Venous gangrene** (phlegmasia alba & cerulea dolens).

N.B: The most dangerous DVT which requires the longest period of treatment is iliofemoral DVT



DD

Venous gangrene

► DD of lower limb edema

A - Unilateral:

I) Acute:

1. DVT
2. Rupture of plantaris m tendon
3. Contusion of calf muscles

➔ [Both previous conditions (No2 & 3) occur during exercise and can produce swollen painful calf which is usually difficult to distinguish from DVT. Duplex scan is needed to establish the diagnosis].

4. Acute filaria

II) Chronic:

- | | |
|-------------------|---------------------|
| 1. Varicose veins | 3. Chronic filarial |
| 2. Cellulites | 4. Lymphedema |

B - Bilateral:

- | | |
|-----------|-----------------|
| ▪ Cardiac | ▪ Nutritional |
| ▪ Renal | ▪ Angioneurotic |

Investigations

DVT is a potentially fatal disease and its clinical picture overlaps with many other conditions. So once clinically suspected, the diagnosis of DVT must be confirmed / excluded by accurate investigations, as clinical examination is not reliable in 50% of cases.

To Detect DVT:

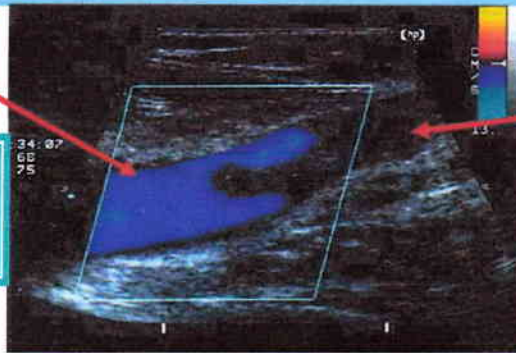
1. Doppler. (accurate in 80-85%)
2. Duplex. (accurate in 90 -100) most errors are in below knee veins
3. Recently radioactive fibrinogen→detect active uptake by thrombus).
4. Recently →spiral CT (most accurate).
5. Venography (not done now) (it is the gold standard but invasive)

Venous blood flow
in the SFV

Fresh thrombus (no
flow) in the SFV with
a floating tail

Colored Duplex US:

Acute DVT of the superficial
femoral vein with a floating tail



If Pulmonary Embolism is suspected:

1. Spiral CT or V/Q lung scan or pulmonary arteriography
2. Ventilation /perfusion scan.
3. Pulmonary angiography.
4. Chest x ray.

If Recurrent DVT in Young Patient:

- Measurement of protein C, S. Antithrombin 3, lupus anticoagulant.

Emboli in veins go to lungs except if there is ASD or
VSD + Eisenmenger → systemic embolization.

Treatment

Treatment

▪ Treatment of DVT

1) Prophylaxis:

- For all patients. - For high risk patients

2) Curative:

- General. - Drug therapy. - Operative.

▪ Treatment of complications:

A. Treatment of DVT:

Prophylactic

For all patients

For high risk patients e.g. major surgery,
elderly patients, history of DVT or PE

- 1- Early ambulation after operation.
- 2- Active leg exercises while in bed.
- 3- Adequate postoperative hydration.

(as for all patients) +

- 4- Elastic stocking support.
- 5- Intra-operative intermittent pneumatic compression.
- 6- Prophylactic anticoagulants e.g., low dose heparin (miniheparin 5000 IU S.C 2 hrs before the operation then every 12 hrs till the pt. is ambulant "5-7 days" this lowers DVT incidence by 50%) or LMWH (at night of operation and 12 hours post operative.

➔ Prophylactic heparinization should be avoided if a large raw area is left after surgery

Curative

A. General:

- Complete bed rest for 7-10 days till the thrombus is adherent to the vessel wall.
- 1st day elastic stockings.
- Vitamin E (helps absorption and recanalization of thrombus).
- Foot elevation in bed 15-20° to decrease edema and pain and increase venous return.
- Thrombi usually takes 7-10 days to become adherent to the vein wall. During this period the patient should be kept in bed then gradual ambulation with elastic stocking support is allowed but standing and sitting with legs dependent are forbidden.
- These measures are continued for 3-6 months until recanalization and collaterization develops.

B. Drug treatment:

► Aim of treatment:

- Prevents formation of new thrombous
- Prevents propagation of thrombus.
- Prevent PE.
- Minimize post-phlebitic limb.

1. Heparin

► Types of heparin:

- 1- Low molecular weight heparin (fractionated heparin):
 - Tinzaparin 175 IU/kg/24 hours S.C.
 - Enoxaparin 1 mg/kg/12 hours S.C.
- 2- Unfractionated heparin is administered either by:
 - Continuous I.V infusion. A loading dose of 80 IU/kg then maintenance dose of 18 IU/kg/hour.
 - This is the ideal method, but it requires hospitalization and a pump that controls the dose per hour.
 - Bolus therapy. A loading dose of 80 IU/kg then maintenance dose of 70 IU/kg/4 hours.
 - Heparin is given until all signs of active thrombosis disappear which is clinically known by disappearance of pain and tenderness on grasping. This usually take 7-10 days.

2. Oral anticoagulants(coumarin derivatives)

- The most commonly used is warfarin.
- Administration:
 - 1- An initial dose of 10 mg warfarin is followed by 5 mg daily dose.
 - 2- Discontinue heparin after 3 days of overlap treatment.
 - 3- 5 days after the start of therapy and PT should be measured and adjusted to keep it at 30-40%.
 - 4- PC and PT are repeated every 2 weeks and the INR should be between 2-3 times the control value.
 - 5- Oral anticoagulants are given for 3-6 months which is the time needed for recanalization. In some patients who are liable for rethrombosis warfarin is given for life.

3. Fibrinolytic Therapy

(The effect of these drugs is at its best if given in the first 3 days of thrombosis, as after that they have no advantages over heparin)

- **Indications:**
 - Isolated ilio-femoral DVT
 - Impending venous gangrene as phlegmasia cerulea dolens
- **Types:**
 - Urokinase.
 - Streptokinase.
 - Recombinant tissue plasminogen activator (advantages: less allergic and more effective).
- **Action:**
 - Conversion of plasminogen into plasmin, which has a proteolytic effect on fibrin & fibrinogen.
- **Advantages:**
 - ↓ valve damage
 - ↓ CVI to 40-50
- **Complications:**
 - Allergic reactions to streptokinase.
 - Bleeding tendency.
 - Intra cranial hemorrhage.

C. Operative:

1. Intraluminal device (filter) (Greenfield filter or Miles or Spencer)(requires local anesthesia)

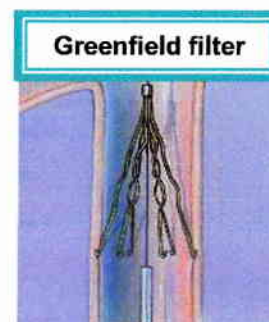
Indications:

- 1- Contraindication for anticoagulants, heparin & fibrinolytics e.g. in hypertension.
- 2- Recurrent pulmonary embolism inspite of full heparinization.

2. Venous thrombectomy (rarely done):

By Fogerty catheter if:

- Massive iliofemoral thrombosis.
- Phlegmasia cerulea dolens.
- Pulmonary embolism.

**B. Treatment of Complications:**

- **Pulmonary embolism:** morphia, O₂, thrombolytics, anticoagulants, embolectomy.
- **Post phlebitic limb:** compression bandage.

More details

Heparin

- It is an acid mucopolysaccharide.
- **Mode of action:**
 - 1- Enhances the activity of naturally occurring antithrombin III.
 - The antithrombin III-heparin complex neutralizes the effect of thrombin.
 - 2- Neutralizes factors IX, X and XI.
 - 3- May have mild thrombolytic effect as it can cause mild activation of fibrinolysis.

	Unfractionated heparin	LMW heparin
Mode of action	Antithrombin III	Anti Xa
Half life	1.5-2 hours	12 hours
Route of administration	I.V	S.C
Monitoring	APTT	Not required- if needed →level of Xa
Incidence of heparin induced thrombocytopenia	1-5%	<2%
	The patient should be hospitalized	May be taken at home

- **Control:**
 - The dose of heparin is monitored by measuring APTT which should be kept between 2-2.5 times the control value.
- **Complications:**
 - 1- Bleeding due to over dosage. It manifests by subcutaneous bruises, epistaxis, bleeding gums or G.I.T bleeding. The antidote of heparin is protamine sulphate 1 mg IV for every 100 IU of heparin. The use of S.C low molecular weight heparin is less likely to cause bleeding.
 - 2- Failure to respond to heparin (heparin resistance). It may be mild and requires only increasing the dose of heparin.
 - 3- Heparin induced thrombocytopenia occurs usually in the 2nd week of therapy. If the platelet count drops below 100000/mm³, heparin should be stopped.

Oral anti-coagulants

- **Mode of action:**
 - These drugs blocks the synthesis of at least 4 vitamin k dependant clotting factors (prothrombin and factors VII, IX and X).
 - For this effect its anticoagulant effect is delayed. Oral anticoagulants inhibit the synthesis of protein S and C which has an anticoagulant effect and so there is a period of relative hypercoagulability during the 1st few days of warfarin therapy. This period should be covered by continuing heparin therapy together with the warfarin for the last 3 days.
- **Complications:**
 - 1- Bleeding is the main problem. It is treated by lowering the dose and by vitamin K injection in a dose of 10-20 mg.
 - 2- Interaction may occur between oral anticoagulants and other drugs as aspirin, barbiturates and H2 blockers.
- **Contraindications:**
 - A- **Absolute:**
 - 1- Trauma or recent operation on the brain or the spinal cord.
 - 2- Bleeding tendencies.
 - B- **Relative:**
 - 1- Major visceral injury.
 - 2- Major acute fractures.
 - 3- History of cerebral hemorrhage.
 - 4- Blood pressure above 180/120 mm Hg.
 - 5- Peptic ulcer.

Axillary & Subclavian DVT

Incidence

- 2-3 % of all cases of DVT

Etiology

- 1- 1ry spontaneous (usually preceded by unusual muscle activity)
- 2- Secondary to:
 - a- Central venous catheter
 - b- Metastatic tumor (axilla)
 - c- Chemotherapy (IV)

Clinical Picture

- 1- Pain and swelling of entire upper extremity
- 2- Fullness of infraclavicular fossa
- 3- Prominent venous collaterals over the shoulder and anterior chest wall
- 4- The limb may be cyanosed

Treatment

As in DVT of the lower limb +

If the vein is compressed at the thoracic outlet, resection of the first rib or scalenus anterior muscle may be required.

Oral Anticoagulants

Mechanism

- Block the synthesis of vit. K dependant factors II, IIV, IX, X
- Prevents synthesis of protein C and protein S which have anticoagulant effect

Administration

- Basal levels of PT and Prothrombin concentration (PC) are estimated before the start of warfarin therapy then measured again 5 days after treatment then every 2-3 weeks

Dose

- Initial dose: 10 mg followed by 5 mg daily dose.
- Given together with heparin for 2 days → then stop heparin (3 times /day → then twice then once / day. Till INR $2\frac{1}{2}$ - $3\frac{1}{2}$ Control PT of the patient

$$\text{INR} = \frac{\text{PT of the patient}}{\text{Normal PT}}$$

Duration

- 3-6 months
- In some recurrent cases give it independently

Antidote

- Vit. K = 10 – 20 mg IV
- Fresh blood, FFB

Contraindications of anticoagulant therapy

A. Absolute

- 1- Trauma or recent operation
- 2- Hemorrhagic diathesis
- 3- Septic phlebitis
- 4- Bacterial endocarditis

B. Relative

- 1- Major visceral injury
- 2- Major acute fracture
- 3- History of cerebral hemorrhage
- 4- Peptic ulcer
- 5- HTN, Bp > 180 / 120

Complications of oral anticoagulants

1. Bleeding is the main problem treated by vit. K.
2. Interaction with aspirin and non-steroidal inflammatory drug
3. Skin necrosis.

Heparin

	Heparin	LMWH
	Is an acid mucopolysaccharide	
Mechanism	- Enhances activity of antithrombin III, antithrombin III / heparin complex - Neutralized factor IX, X, XI - Mild thrombolytic effect	Home TTT ↓ bleeding No lab. Test to control dose
Duration	Given till all symptoms and signs of active thrombolysis subsides (7-10 days) Or Till oral anticoagulants starts to work (INR = 2)	
Dose	50-100 IU / kg 1000-2000 IU / H a- Continuous IV infusion b-Bolus therapy / 4-6 hours c-SC method (calciparin) / every 12 hours	
Control	a PTT 1 ½ : 2 ½ control value	

Complications of heparin therapy

- 1- **Bleeding** due to over dose manifested by epistaxis, subcutaneous bruises, hematuria etc...
 - **Management:**
 - proper dose control by APTT
 - Use of antidote protamine sulfate (1 mg IV per 100 IU heparin)
- 2- **Heparin resistance:**
 - **Management:** • by ↑ the dose
- 3- Thrombocytopenia
- 4- Osteoporosis

Pulmonary Embolism (PE)

Incidence

- 2-3% of all hospital mortalities are due, wholly or in part, to pulmonary embolism.

Causes

- Usually secondary to DVT.
- Infective endocarditis.
- Other emboli: fat, air, infected, tumor embolism.

Clinical Picture (differs according to the size of embolus)

1. Small emboli (impacted in peripheral arterioles).

- Usually silent.
- Recurrent small emboli will lead to pulmonary hypertension.

2. Medium-sized emboli (impacted in branches of pulmonary artery):

- Leads to pulmonary infarction (less than 10% of the cases)
- This leads to classical picture of:

a. Severe pleuritic pain.

b. Dyspnea.

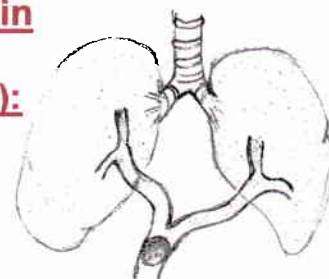
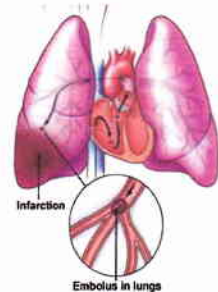
c. Hemoptysis.

3. Large-sized emboli (impacted either in the main pulmonary artery itself or in one of its major branches: (pulmonary artery occlusion > 50%):

- This leads to massive pulmonary embolism.

▪ Clinical picture:

- a- Severe pericardial pain.
- b- Tightness in chest.
- c- Marked dyspnea.
- d- Severe hypotension.
- e- Marked tachycardia.
- f- Sudden death may occur.



Fatal pulmonary embolism

- It should be noted that in many patients with pulmonary embolus, there is no clinical evidence of DVT in the lower limbs.
- Unexplained dyspnea or heart failure in a hospitalized patient is very suggestive of pulmonary embolism.

DD of Acute Chest Pain + Shock

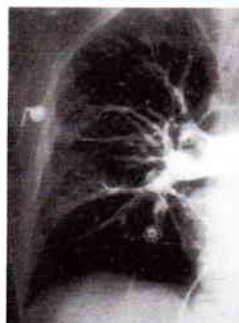
- Tension pneumothorax, acute myocardial infarction, cardiac tamponade.

Investigations

1. **Spiral CT (recent):** most accurate. The clot appears as a filling defect
2. **Ventilation Perfusion test:**
 - By ventilation & then injection of isotope material.
 - Shows filling defect in perfusion scan with normal ventilation scan (in fibrosis → defect in both ventilation & perfusion scan).
3. **Blood tests**
 - ↑ LDH & serum alkaline phosphatase & normal bilirubin (↑ in case of heart failure).
 - **Blood gases:** hypoxia ($\text{PaO}_2 < 80 \text{ mmHg}$, PaCO_2 remains normal) while in fibrosis there is ↑ PCO_2 .
4. **ECG** → P-pulmonale. Axis deviation to the right, right ventricular strain pattern (in 40% of cases).

5. CXR:

- May be normal (in 50% of cases).
- Wedge shaped peripheral opacity is rare.
- ↑ or ↓ vascularity.
- Pleural effusion.



6. Pulmonary angiography

- Is the most accurate, yet not commonly done (invasive).
- Can show hypoperfusion.

Start with D-dimer → if +ve → continue investigations
→ if -ve → Stop

Treatment

Prophylaxis & TTT of DVT is very important in prevention of PE (mention)

☞ For Pulmonary Embolism:

a. Curative:

- Small and medium-sized pulmonary embolism are treated by anticoagulants.
- Patients with massive pulmonary embolism (surgical emergency):
 - Cardiac catheterization (in pulmonary artery)
 - Thrombolytics e.g. streptokinase 600,000 unit followed by 100,000 unit / hour for 72 hours.
 - The cardiac functions should improve within 6 hours in favorable cases, if failed → urgent pulmonary embolectomy is performed.

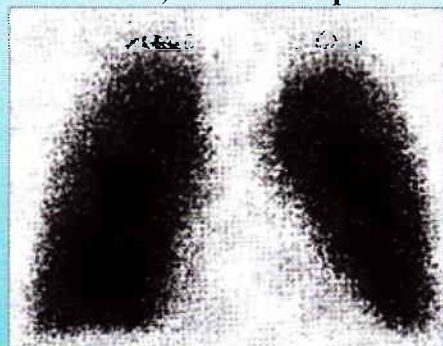
b. In recurrent cases:

- Prophylaxis against recurrence of pulmonary embolism by IVC filter.

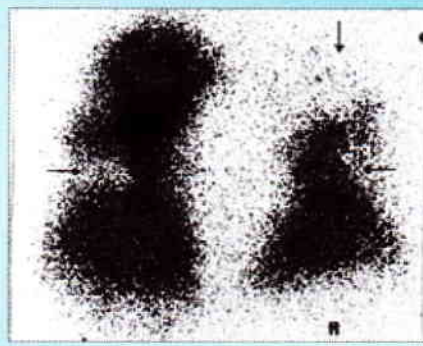
Ventilation – perfusion scan (V/Q scan): is a radioisotope scan that can measure and compare the amount of blood flow to each lung (perfusion) and the amount of air that goes to each lung (ventilation)

In pulmonary embolism, the perfusion of a segment of the lung is impaired (*because the embolus occludes one of the branches of the pulmonary artery*) while the ventilation of the same segment is normal.

That is called, **ventilation – perfusion mismatch**



Normal pulmonary ventilation



Abnormal pulmonary perfusion

Special types of pulmonary embolism

1. Septic PE:

Source

- Septic thrombophlebitis

Size

- Usually small → multiple foci of infection in the lung.

Treatment

- Proximal venous ligation.
- Antibiotic in high doses.
- Drainage of any closed site of infection.

2. Bone marrow fat emboli:

- It is a rare condition.
- 95% of fat emboli are associated with fractures which disrupt venous channels in the bone marrow, allowing fat particles to enter the venous circulation.

Clinical picture

- May be asymptomatic or
 - Dyspnea.
 - Tachypnea.
 - Cerebral symptoms (as confusion, delirium or coma).
 - Petechial hemorrhage on chest wall, axilla & flanks
 - Cyanosis.
 - Pleural rub (12-96 hours after injury).

Varicose Veins

Definition

☞ Clinical Definition:

Dilated, elongated, and tortuous superficial veins of the LL.

☞ Pathological Definition:

Veins that allow reversal of blood flow through damaged valves).

- ▶ 1ry (85%), 2ry (15%)
- ▶ Pain, Disfigurement
- ▶ **Comp.:** Venous, Skin
- ▶ **Invest.:** Doppler, Duplex, comp.
- ▶ **TTT:** Conservative, Sclerotherapy, Surgery

Etiology & Types

A- Primary (85%): no apparent cause, may be due to:

1. Congenital mesenchymal weakness (as Marphan, Ehlar danlos, Askar syndrome):
 - Weakness of the wall of the veins → venous dilatation even with normal pressure → valvular incompetence
2. Congenital valvular incompetence.

⇒ Aggravating factors:

- Female sex, occupation with prolonged standing, high parity, marked obesity, positive family history in 50% of cases and OCP.

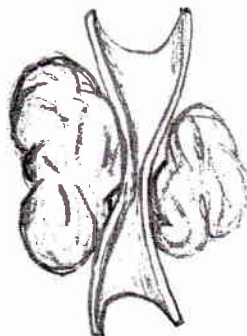
B- Secondary (15%)

1. DVT (commonest cause)
 - Recanalization of the thrombus → damage of the valve → reflux of blood → this postulates an unusual strain on the superficial veins which have little external support → they progressively dilate.

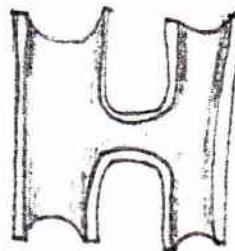
2. Pelvic tumors (fibroid) and pregnancy.
 - Due to compression of the pelvic veins.
 - Also hormones of pregnancy e.g. progesterone and relaxin action on veins.
3. A-V fistula (arterial pressure is transmitted to deep system leading to valvular incompetence)
 - a- Congenital: local gigantism (Klippel-Trenaunay syndrome), Parkes Weber Syndrome (collection of varicose veins secondary to microscopic A-V fistula)
 - b- Acquired: butcher's thigh.
4. Aneurysm.
5. Buerger's disease.



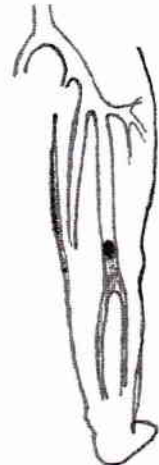
Pregnancy



Compression by tumor



A-V fistula

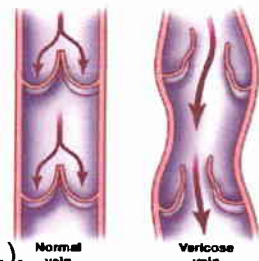


DVT

Pathology

Site

- a. If primary → in perforators and superficial system only.
- b. If secondary → in perforators, superficial and deep systems.



Macroscopic Picture

1. **Tubular type:** → dilated main veins (common in 1ry V.V.).
2. **Spider type:** → veins are thin & blue (dilated skin venules).
3. **Serpentine type:** → veins are coiled.
4. **Saccular type:**
 - Blow out dilated superficial system in front of incompetent perforators.
 - Palpable defect in the deep fascia opposite it.
 - Saphena Varix is a sacculle opposite an incompetent saphenofemoral junction.



Serpentine



Saccular



Tubular

Microscopic Picture

- The wall is thickened.
 - The muscle & elastic layers are replaced by fibrous tissue.
- Above & below knee perforators can be called communicators as they join the long saphenous directly, while the ankle & lateral perforators join branches of the long & short saphenous respectively.
 - **Communicators** = direct perforators.
 - **Perforators** = indirect perforators.
 - **Factors that help venous return from lower limb:**
 - The muscle pump (soleus acts as peripheral heart).
 - The uni-directional valves.
 - Negative intra-thoracic pressure.
 - Transmitted arterial pulsations to venae comitans.

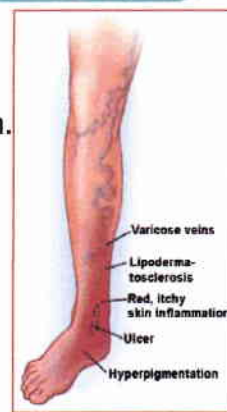
Complications (due to ↑ venous pressure)

Venous Complications

1. **Superficial thrombophlebitis:** due to sluggish circulation.
2. **Hemorrhage** after trauma (profuse) due to ↑ pressure.
3. **Edema of the LL:** due to ↑ venous pressure.

Skin Complications

- 1- **Pigmentation, Dermatitis & Eczema:**
 - Due to ↑ venous pressure → capillary dilatation → RBCs get out in the S.C fat → phagocytosed → hemosiderin in SC tissue → pigmentation & irritation → eczema.
- 2- **Varicose Ulcer:** see later.
- 3- **Lipodermatosclerosis:**
 - Pigmented woody induration of the leg associated with dull aching or bursting pain
- 4- **Malignancy:**
 - On the top of varicose ulcer → Marjolin ulcer.
- 5- **Talipes Equinus:**
 - Extremely rare occurs after long standing varicose ulcer.
 - (Walking on toes relieves the pain → shortening of tendo-achillis).



From 1 → 6 together with chronic leg pain constitute **post-phlebotic syndrome** as they usually follow previous DVT with incompetent perforators.

Clinical Picture

Type of patient

- Primary V.V usually occurs around 30 years, rare < 20 years.

Symptoms (May be asymptomatic)

1. **Cosmetic disfigurement.**
2. **Aching and discomfort:** of the limb usually described as dull-aching, heavy, bursting sensation with sense of hotness usually at the end of the day or on prolonged standing and is relieved by elevation of the limb.
3. **Night cramps:** due to congestion of the muscles (in severe cases).
4. **Mild swelling:** occurs at the end of the day (mainly with secondary)
5. **Symptoms of complications:** e.g. pigmentation, itching (common), ulceration, etc....

Signs

A. General:

- 1- Pulse and BP changes in cases of A-V fistula.
- 2- Heart examination for signs of peripheral of A-V fistula.
- 3- Flat foot and kyphosis (signs of mesenchymal weakness)

B. Abdominal:

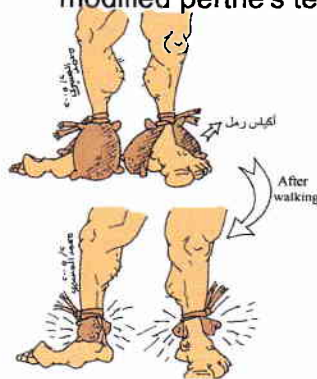
- Scars of previous operation, dilated veins crossing inguinal region, abdominal or pelvic swelling.

C. Local (while patient is standing):

- 1- Inspection: LL is inspected for:
 - a- Varicosity and its anatomical site.
 - b- Any swelling (saphena varix), scar, edema.
 - c- Skin complications: e.g ulcer, pigmentation.
 - d- Corona phlebectatica: ankle / maleolar flare (one of the earliest manifestations of CVI in form of dilated subdermal veins at or just below the medial maleolus with thin fragile overlying skin → blue bleb appearance.
- 2- Palpation:
 - a- Thrill on cough at saphena varix or incompetent saphenopopliteal junction but may be spontaneous in case of A-v fistula.
 - b- Fascial defect: felt at the site of incompetent perforator (Fegan sign)
 - c- Superficial thrombophlebitis: firm tender nodule.
 - d- Skin temperature and peripheral arterial pulsation to exclude associated arterial disease.

NB. Prominent varices may be tender particularly in menstruating females

- 3- Percussion: Schwartz's test (tapping test) to detect:
 - Dilated veins belong to short or long saphenous.
 - Denotes incompetent valves in superficial system
- 4- Auscultation: for machinery murmur (A-V fistula)
- 5- DRE or PV: for pelvic tumors.
- 6- Special test:
 - For localization of incompetent perforator (Trendlenburg's test, multiple tourniquet test)
 - Differentiate between occluded and patent deep systems (Perthe's , modified perthe's test)



Multiple dilated, elongated, tortuous veins of the LL

Differences between primary and secondary V.V

	Primary V.V	Secondary V.V
Etiology	Idiopathic	Secondary to DVT, A-V fistula, pelvic tumors or pregnancy
Pain	Slight or absent	Marked
Distribution	Along the long or short saphenous veins	Haphazard veins crossing the groin may be seen
Complications	Minimal or absent	More common

Investigations

The aim of investigations is detection of sites of incompetent perforators and to verify the patency of deep system. In most cases of 1ry V.V clinical assessment is sufficient.

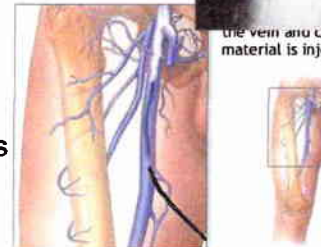
For Varicose Veins

- **Doppler U/S, Duplex:** it shows:
 - Reversal of blood flow.
 - Venous thrombosis.
 - Dilated tortuous veins.
 - Incompetent valves.
- **Venography** → (rarely done).
 - Ascending: exclude DVT.
 - Descending: shows incompetent veins (presence of valves & any incompetent perforator).



For Complications

- **Arteriography:** in case of arteriovenous fistula.
- **Plain X-ray:** in case of varicose ulcer → if periostitis is suspected.
- **Biopsy:** if malignancy is suspected.



DD

1. Other causes of leg pain.
2. Other causes of leg swelling.
3. Other causes of leg ulcers.

Treatment

1. Primary V.V.

A. Conservative treatment:Indications:

- Early uncomplicated 1ry V.V.
- Patient unfit for operation or refusing operation.

Method:

1. Reassurance.
2. Removal of any predisposing factor.
3. Compression therapy: this is done by graduated elastic compression stockings which exerts 40 mmHg at the ankle and 20 mmHg just below knee (this retards the progression of CVI)
4. Elevation of the LL to prevent congestion.
5. Vitamins, antioxidants, veno-tonics.

Treatment

▪ **Of V.V:**a. Primary:

1. Conservative.
2. Injection.
3. Surgery:
 - Trendlenburg
 - SC stripping.
 - Triple ligation.
 - Recent lines.

b. Secondary.▪ **Of complications.**

B. Injection (Compression Sclerotherapy):

Indications:

1. Moderate cases of 1ry V.V. with main complaint of cosmetic disfigurement.
2. Residual V.V after operation.
3. Lower leg perforators.
4. Recurrent V.V.



If the main complaint is pain, injection will not cause its relief because the pressure is still high, so we should refer to surgery.

Complications:

1. Syncope due to drug sensitivity.
2. Extravasation leading to sloughing & necrosis of the overlying skin.
3. DVT (may occur if large amount of sclerosant material reaches the deep system undiluted).

Contraindications:

- Septic thrombophlebitis, Secondary V.V.
- Avoid injection of long Saphenous itself.

Microsclerotherapy: thread veins and reticular varices may be treated by injection through a very fine needle; very dilute sclerosing solutions are used. This technique is referred to as microsclerotherapy.

Foamsclerotherapy: sclerosing solution is forcibly mixed with air to create a foam agent often used for large diameter vessel

C. Surgical treatment:

1. Indications:

- Large 1ry V.V.
- Presence of complication.

2. Pre-op:

- Localization of veins & incompetent perforators and marking their site.

3. Postoperative:

- Compression bandage is applied to the limb at the end of the operation to prevent excessive bruising.

4. Complications of surgery:

- a- Bruises and discomfort.
- b- Pain (requires only mild analgesic)
- c- Sensory nerve injury may occur (1% of operation).
- d- Motor nerve injury (uncommon).
- e- Venous thrombosis is often seen in residual varices following surgery.

2. 2ry V.V

- A.** Post phlebitic (i.e. following DVT). The majority of these cases are treated conservatively by elastic stockings. Rarely the varicosities are large enough to require active treatment. In these cases verify that the deep system is patent by Duplex, and then treat as 1ry V.V.
- B.** - V.V complicating A-V fistula. Surgical treatment of the fistula is usually followed by marked regression of the varicosities.
- If residual veins remain, treat as 1ry V.V.
- C.** V.V occurring during pregnancy. A complete elastic stocking from the toes up to the groin is applied all through the period of pregnancy. After labour any residual V.V are treated as 1ry V.V.

3. Management of Complications

Pigmentation & eczema:

- i) Local cleanliness & avoid itching.
- ii) Rest & elevation.
- iii) Hydrocortisone & zinc oxide ointment.

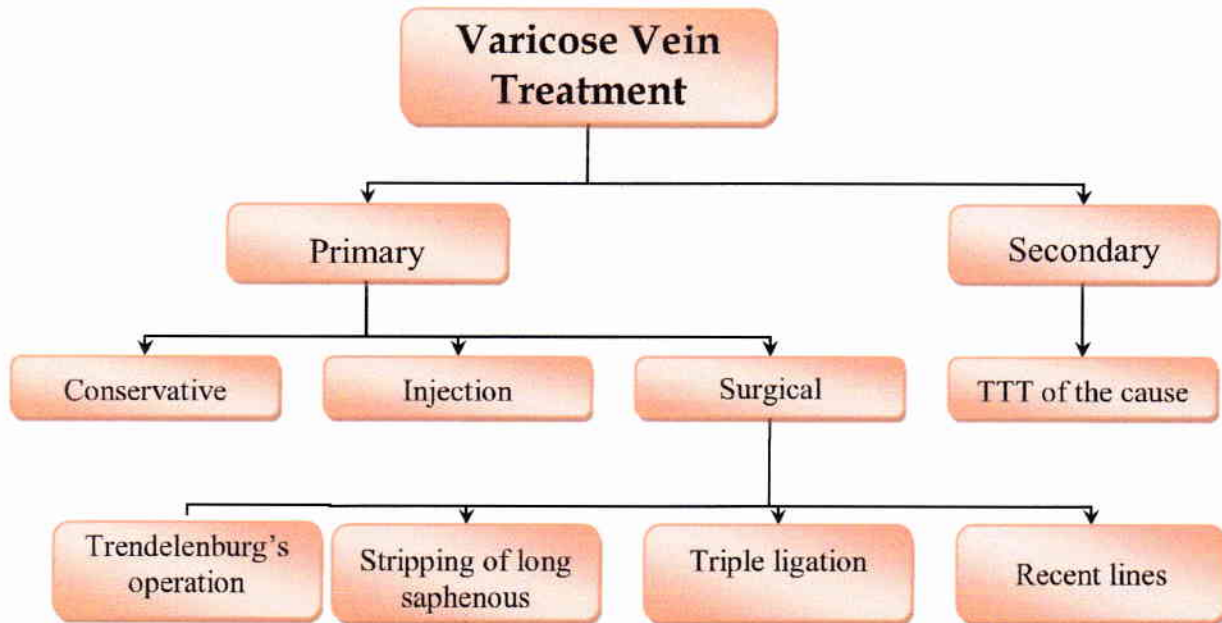
Varicose ulcer → see below

Hemorrhage → elevation & pressure bandage.

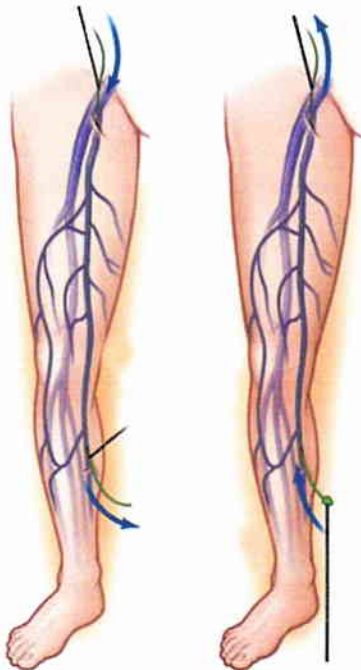
Marjolin's ulcer → excision by safety margin & cross leg flap.

Talipes equines → physiotherapy.

Periostitis → saucerization.

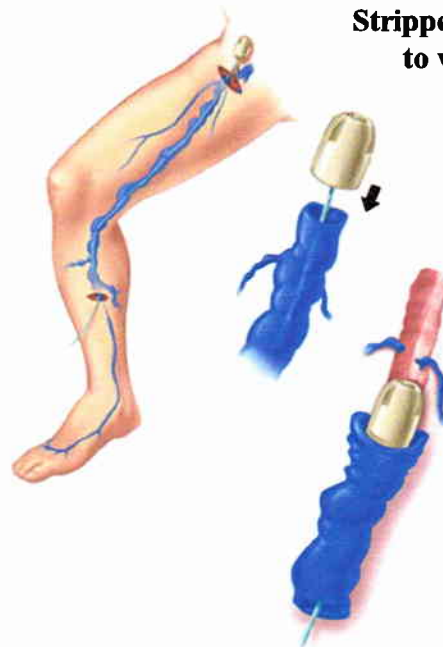


**Vein stripper enters
groin incision**

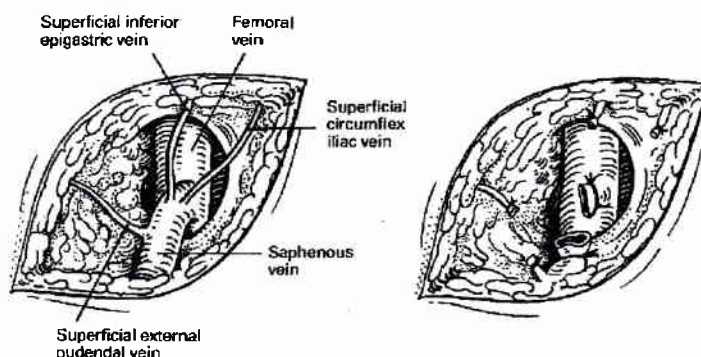


**Vein stripper exits calf
incision**

**Vein stripper pulled through
leg removes diseased vessel**



**Stripper head attached
to vein stripper**



More Details

1. Injection sclerotherapy:

► Method:

- The patient lies supine.
- The leg is hanged down beside the bed, so the veins will be distended & the needle for injection is inserted inside the vein.
- Then the leg is elevated on the bed & the vein is compressed by the index & middle fingers which are applied around the needle.
- The sclerosing solution is injected into the empty vein.
- A firm bandage is applied to allow fibrosis of the intima.
- The patient is asked to walk to allow the blood to dilute the irritant substance to avoid blocking of the deep system.
- The interval of injection: every 4 weeks.

► Material used:

- The commonest sclerosing material used is ethanolamine oleate 5% (bad cosmetics) which damages the vein intima, so that a septic thrombosis and later on fibrosis will occur. The dose injected should not exceed 2ml per site, since larger doses will easily reach the deep veins and cause thrombosis.

► Effect:

- The injected material causes irritation of the endothelium & obliteration of the veins by fibrosis.

Safe and effective Sclerotherapy

- 1- Veins that should not be injected:
 - Lower third of the leg
 - At ankle
 - On foot
 - Infant legs
 - In post-phlebitic stasis dermatitis
- 2- No more than 0.5 ml of the sclerosant sodium tetradecyl sulfate 3%
 - Hypertonic saline
 - Morrhuate Saline
- 3- Injection should be done during the patient is reclining not standing
- 4- Compress above and below the injection site for 1 min.
- 5- A compressive elastic bandage is applied and the patient is actively ambulated immediately

CEAP Classification of Varicose Veins

C stands for Clinical classification

- Co No visible or palpable signs of venous disease.
- C1 Telangiectasis or reticular vein. Veins less than 3 mm
- C2 Varicose veins. Veins over 3 mm
- C3 Edema
- C4 Skin and subcutaneous changes

Now divided to better define severity:

- C4a Pigmentation or eczema
- C4b Lipodermatosclerosis or atrophie blanche
- C5 Healed venous ulcer
- C6 Active venous ulcer

Further descriptors "a" for asymptomatic, "s" symptomatic

E stands for Etiology Ec: Congenital

- Ep: Primary
- Es: Secondary (post-thrombotic)
- En: No venous cause identified

A stands for Anatomy

- As: Superficial veins
- Ap: Perforator veins
- Ad: Deep veins
- An: No location identified

P stands for Pathophysiology

- Pr: Reflux
- Po: Obstruction
- Pr,o: Reflux and obstruction
- Pn: No pathophysiology identifiable

Injection Sclerotherapy

► Method:

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► Effect:

- The injected material causes irritation of the endothelium & obliteration of the veins by fibrosis.

6-Abd Swelling:	- Splenomegaly, Para-aortic L.N., intestinal Obstruction & obstructive jaundice.	
Classification	(Ann Arbor classification) Stage I → Enlarged 1 group of LN. above or below diaphragm. Stage II → Enlarged more > 1 group of LN. above or below diaphragm Stage III → Enlarged LN. above & below diaphragm Stage IV → Extranodal affection especially liver & bone marrow	
Investigation	- o For diagnosis → excision biopsy if L.N. is involved. - o For D.D. & staging: a. CBC (pancytopenia , & ESR > 100) b. LFT (↑billirubin due to obstructive, HC or hemolytic) c. KFT (↑ uric acid due to tumor lysis \$) d. CXR. e. Abdominal U/S & C.T. f. BM puncture. g. Staging laparotomy is rarely used (replaced by C.T).	
Treatment	1. General →Vitamins, blood transfusion, iron therapy. 2. Definitive treatment <u>Stage I, II & IIIa</u> → radiotherapy a- If above diaphragm →Mantle. b- If below diaphragm →Inverted Y. c- If IIIa→Both mantle & inverted Y <u>Stage III b & IV</u> → Chemotherapy. MOPP (Mustine, Oncovine, Prednisone & Procarbazine)	(mainly chemotherapy as it is multicentric) As Hodgkin's lymphoma but: <u>In nodular type</u> → chlorambucil <u>In diffuse type</u> → cyclophosphamide, Vincristine & Prednisone.

Chronic Leg Ulcers

1. Chronic Traumatic Ulcers

- It is first produced by trauma then it becomes chronic from negligence and repeated infection.
- Finally it becomes fixed to the bone and this prevents contraction of the base of the ulcer and interferes with the growth of the epithelium.

Causes: Wounds, burns, radiation, bedsores.

Characters:

- Number: Usually single.
- Site: Skin over medial side of tibia or bony prominence.
- Edges: Punched out.
- Floor: Healthy or unhealthy granulations.
- Base: Indurated.
- Margin: Pigmentation or inflammation.
- Discharge: Serous or purulent.
- Inguinal L.N.: Firm, tender, mobile.

Treatment:

- Conservative: Like varicose ulcer.
- Surgery: Cover with skin graft or flap.

2. Inflammatory Ulcers

- a. **Chronic osteomyelitis ulcer**: See orthopedics.
- b. **Chronic specific ulcers**: Rare, T.B., Syphilis, Madura foot discharge sulphur granules.

Syphilitic Ulcer:

- It is rare nowadays.
- Site: It occurs on the medial surface of the upper third of the tibia.
- Number: It may be solitary or multiple.
- Edge: is punched out and may be serpiginous.
- Floor: is covered with yellowish (wash leather) slough.
- Base: may be fixed to bone.
- W.R is positive.



Tuberculous Ulcer:

- It results from the breaking out of a tuberculous arthritis or osteitis.
- Edge: thin undermined
- Margin: bluish.
- Floor: pale granulation tissue
- Base: Slight induration



3. Neoplastic Ulcers

- Primary skin tumors as sq. cell carcinoma, melanoma
- Marjolin's ulcer on top of chronic benign ulcer.
- Ulcerating deep malignancy as osteosarcoma, fibrosarcoma

1. Squamous-cell carcinoma or epithelioma:

- It may occur de novo in the skin or on the top of a chronic scar when it is known as Marjolin's ulcer.
- Premalignant conditions include chronic venous ulcer and prolonged irritation of the skin by chemicals e.g. dyes, tar or soot.
- Shape is irregular in outline.
- Edges are raised and everted.



Lymph Nodes Enlargement

Localized

1. Acute septic lymphadenitis
2. Chronic lymphadenitis
3. TB lymphadenitis (caseous)
4. \$ (first stage).
5. Metastasis.
6. Early lymphomas
7. Lymphogranuloma inguinale
8. Filarial lymphadenitis

Generalized

1. Leukemia
2. Late lymphomas
3. TB lymphadenitis (fibroid) .
4. Glandular fever

DD of TB Lymphadenitis

According to the clinical presentation

For lymphadenoid type

Chronic non specific type

- No tuberculous toxemia, no caseation, no sinus.
- There is a septic focus.
- Small & equal L.Ns.

Hodgkin's lymphoma

- Progressive course.
- Rubbery.
- Spleen may be palpable.
- Pruritis

For lymphatic borne

Secondaries in the LNs.

- Short progressive course.
- L.Ns. hard, fixed.
- No calcification.
- The primary lesion.

Cold abscess from other swelling in the neck.

Sinus from other sinuses in the neck as thyroglossal, branchial, actinomycosis.

- Base is indurated and soon becomes attached to the deeper structures.
- Discharge: A blood-stained occurs and increases if there is superadded secondary infection.
- Inguinal lymph nodes are first not palpable but later they become involved and become hard.

2- Malignant melanoma: (see general)

- It usually affects adults.
- It may start de novo as a pigmented (dark brown or black) lesion, which grows progressively with a tendency to ulcerate and bleeding.
- Sometimes it takes the form of a rapidly growing, fleshy, ulcerated skin tumor (amelanotic melanoma).
- Satellite nodules may be present around the tumor (due to spread by lymphatic permeation).
- Inguinal lymph nodes are first not involved but later become palpable & hard.
- Blood borne metastases may be seen in liver & lungs in advanced cases.

4. Vascular Ulcers

	(A) Ischemic Ulcer	(B) Venous Ulcer
Site	Between the toes, in the dorsum of the foot or around the malleoli	In the ulcer bearing area(Gaiter area) just above the medial malleolus
Edge	Punched out	At first → irregular, sloping and serrated then → punched out
Margin	Hyperemic	Skin around the ulcer is pigmented
Floor	Granulation or pus	Infected ulcer → dirty granulation tissue Non infected ulcer → healthy granulation tissue
Base	Difficult to be palpated	Indurated(hard)

(C) Lymphoedema Ulcer: from rupture of an infected bulla.

5. Neurotrophic Ulcers

Cause: skin anesthesia in some neurological diseases e.g. peripheral neuritis, nerve injuries or DM.

Site: sole of the foot at ball of the big toe or heel (pressure areas).

Pathology:

- Skin anesthesia → trauma not felt by the patient → callosity & adventitious bursae → infection of bursae → separation → deep penetrating ulcer to bone & joints ... **IT'S PAINLESS.**

Treatment:

- Conservative treatment + control neurologic disease.
- Amputation if destroyed bone or joints.

6. Blood Diseases

As sickle cell anemia & congenital spherocytosis.

7. Autoimmune Disorders

SLE, rheumatoid arthritis.

Venous Ulcer (Varicose Ulcer)

"Most common cause of leg ulcer"

Definition

- Discontinuity of epithelium of the skin resulting in a 3D conical defect with well formed edge, margin, floor and base in relation to high venous pressure of the lower limbs.

Incidence

- Constitutes 90% of chronic leg ulcers.

Causes

- Usually post phlebitic limb due to DVT.
- Less common due to A-V fistula.
- Rarely due to primary varicose veins.

Pathogenesis

- Recanalization of DVT is commonly followed by dysfunction of the valves of both deep veins and the perforating veins.
- As the skin of the lower part of the leg is drained directly by the perforators which drain into the deep system, it follows that incompetence of these perforators leads to high venous pressure in the skin particularly that lying on the medial aspect of the lower third of the leg (ulcer bearing area).
- Ambulatory venous hypertension → skin capillaries become glomerulous-like → trapping of more leucocytes which become activated and release proteolytic enzymes that damage the capillaries → fibrotic process in skin and subcutaneous tissue (lipodermatosclerosis).
- The combination of venous hypertension, eczema and liposclerosis will eventually lead to ulceration after minor trauma.
- Local hypoxia due to venous stasis and impaired nutrition lead to the liberation of free oxygen radicals which are toxic to the tissues.

Clinical Picture

👉 **Clinical picture of the cause (see before)**

In 50% of cases there are no manifestations of varicose veins, the ulcer is due to incompetent ankle perforators only.

👉 **Ulcer of the following criteria**

Site → in the ulcer bearing area (Gaiter area) just above the medial malleolus.

Number → usually solitary.

Size

- Variable.
- In severe cases, it may encircle lower part of leg above ankle.

Floor

- Infected ulcer → dirty granulation tissue.
- Non-infected ulcer → healthy granulation tissue.

Base → Indurated (hard).

Edge →

- At first → irregular, slopping & serrated then → punched out.

Margin → the skin around the ulcer is pigmented and may show contact dermatitis.

LN's → +ve if 2ry infection or malignant changes.

Complications

- Infection, hemorrhage, osteomyelitis, peritonitis, marjolin ulcer, talipes equinovarus.

DD of Chronic Leg Ulcer

Mention:

- **Ischemic ulcer:** at pressure sites, deep, painful + ischemic changes of L.L.
- **Neurotrophic ulcer:** painless, deep, penetrating to bone, at pressure sites.
- **Malignant ulcers:** everted edge, necrotic floor, indurated base.
- **TB ulcer:** undermined edge, cyanotic margin, caseous material.
- **Syphilitic ulcer:** punched out edge, wash leather floor, WR +ve.
- **Chronic traumatic ulcer:** punched out, over bony prominence, LNs firm, tender, mobile.

Investigations

- For the cause e.g.: Doppler, duplex (functional & anatomical), biopsy if suspecting malignancy.

Treatment

■ Treatment of Ulcer

i. Conservative treatment for early ulcer (main line of treatment):

- **Rest.**
- **Elevation** of the limb.
- **Compression stocking** (below knee) → exerts 40 mmHg pressure at the ankle and 20 mmHg below knee. It is the most important item in treatment.
- **Dressing** with saline & not antiseptics because of eczema. Most ulcers heal in 3-4 weeks.
- **Debridement:** removal of any dead tissue on the wound is essential for proper healing.
- Some **medications** are said to accelerate ulcer healing as:
 - Pentoxifylline.
 - Prostaglandin E1 analouge.
 - Diosmin.
- Conservative treatment is usually successful in allowing the ulcer to heal.
- The problem is that, once the patients return to normal activity, most ulcers will recur.

ii. Surgical For resistant ulcer:

- If there is evidence of incompetent perforators in the leg, they can be either injected or excised.

N.B.: Previous operations as Cockett's or endoscopic subfascial ligation of perforators are not commonly performed nowadays.

iii. Prevention of recurrence

- Continue use of elastic stocking after treatment.
- Leg elevation whenever possible.

Chronic Venous Insufficiency

Definition

- CVI is a chronic disease in which venous return is impaired usually over number of years of
 - Reflux
 - Obstruction
 - Calf muscle failure
- This leads to venous hypertension

Pathophysiology

A- Changes in macrocirculation:

- Failure of
 - Effective calf muscle contraction
 - Vein patency
 - Valvular competence

B- Changes in microcirculation

- 1- Fibrin-cuff theory: widening of pores between endothelial cells allow passage of large molecules out of intra-vascular space into the tissue then fibrin accumulation at this sites around the capillaries forming cuff (barrier to O₂, nutrients) → lack of supply to skin, interstitial tissue → local tissue ischemia, cell death → ulceration
- 2- Cutaneous iron Over load
 - ↑ hydrostatic pressure within capillaries combined with ↑ capillary permeability → extravasation of RBC's leading to:
 - iron promotes production of O₂ free radicals, lipid peroxides → tissue destruction

Risk factors

- Family history of DVT, VV
- Obesity
- Smoking
- Pregnancy
- Jobs requiring long periods of standing or sitting

Causes

- Venous hypertension
- DVT
- Phlebitis
- Congenital absence or weakness of valves in the leg veins

Clinical Picture

Type of patient

- Female > male

With history of (may not be present)

- DVT – PAD – DM

Symptoms and signs

- 1- Ankle, leg edema (1st symptom)
 - typically → edema worse at end of day
 - Improves by leg elevation
- 2- Leg discomfort: heaviness-aching or burning sensation
- 3- V.V
- 4- Lipodermatosclerosis
- 5- Ulcers

Investigations

1) Color coded Duplex Ultrasound scan:

(Salient investigation method)

- Identify deep v. obstruction
- Identify deep vein valve incompetent (Reflux)
- Presence and location of incompetent perforators

2) Air plethysmography (APG):

3) Venography:

- Ascending → vein obstruction
→ Incompetent perforators
- Descending → incompetent D.V valves

4) Manometry:

Measures the pressure in dorsal foot vein in standing & in walking

- Normally: pressure on walking decreases
- CVI → walking venous hypertension

DD

From other causes of leg pain and leg swelling:

1. Vascular:

- Superficial thrombophlebitis.
- Congestive heart failure
- DVT
- Post-phlebitic syndrome.
- Lymphedema.

2. Dermatologic:

- Cellulitis.
- Dermatitis.

3. Endocrinal and metabolic: Liver cell failure.

4. Traumatic: Fracture, muscle tear, hematoma.

Treatment

Mainly conservative

A- Conservative:

- 1- Avoid prolonged standing or sitting
- 2- Frequent leg elevation
- 3- Graduated elastic compression (highest ankle, decreased proximally)
- 4- Dressing
- 5- Systemic medication to improve the healing (Zink, standozolol)

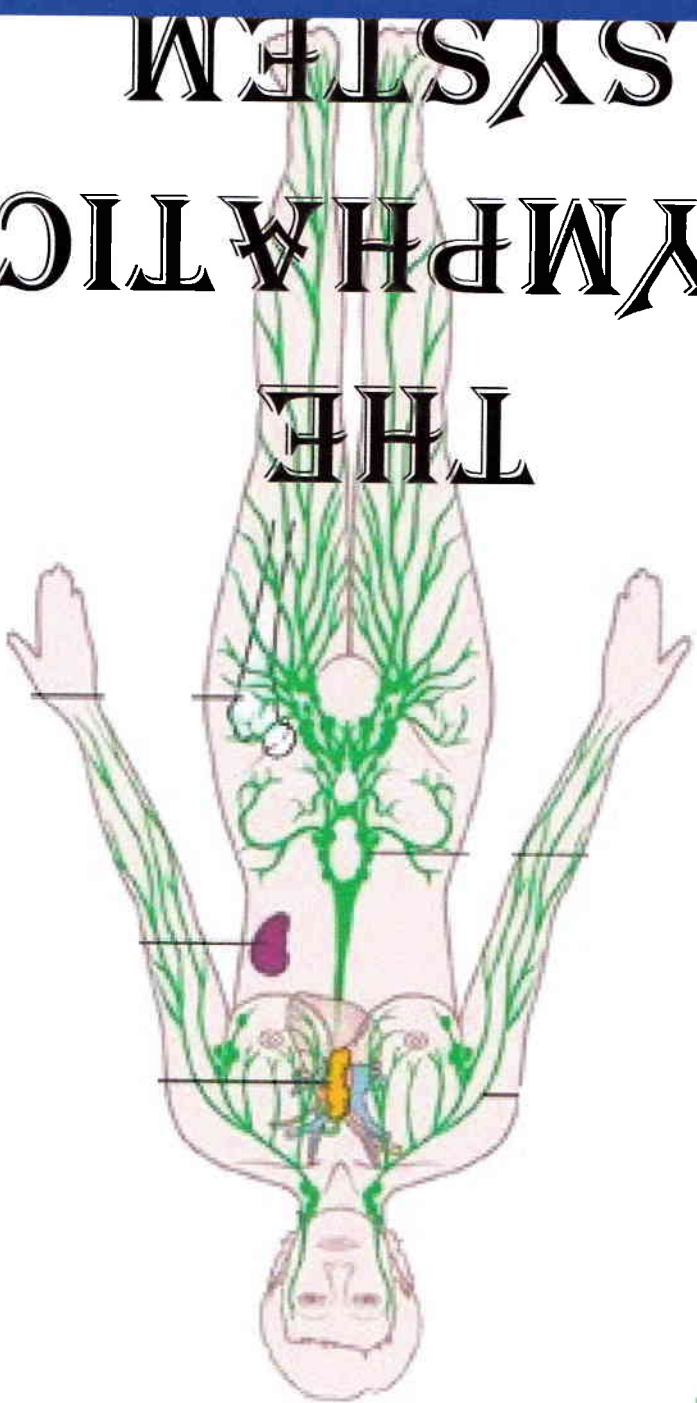
B- Surgical Treatment

- Uncommonly used.
- Results not satisfying.
 - **Procedure:**
 1. Vein stripping.
 2. Bypass surgery.
 3. Valve repair.

Points for MCQ

- ▣ Pressure in superficial veins in the leg is 80 mmHg & it falls during exercise.
- ▣ Acute lipodermatosclerosis is treated by anti-inflammatory drugs & external compression.
- ▣ Acute superficial thrombophlebitis is treated by systemic anti-inflammatory drugs & external compression.
- ▣ Duplex U/S is the accurate non-invasive investigation used to evaluate venous reflux & patency.
- ▣ Phlegmasia alba dolens is due to iliofemoral vein thrombosis + lymphatic obstruction.
- ▣ Treatment of phlegmasia cerulea dolens is nearly always surgical.
- ▣ The commonest cause of fatal pulmonary embolism is iliac vein thrombosis.

THE LYMPHATIC SYSTEM



CHAPTER 3

Lymphedema

Definition

- Accumulation of fluid in the interstitial space due to lymphatic affection with edema of the overlying skin, which becomes thick corrugated.
- Edema at first pitting later on non-pitting.
- May run in families "**Milroy's disease**".
- It affects extremities, scrotum, external genitalia and rarely the breast.

- ▶ 1ry, 2ry
- ▶ C/P: swelling, pain, disability
- ▶ Comp.: infection, elephantiasis, sarcoma
- ▶ TTT: mainly conservative.
- ▶ N.B.: Stemmer's sign

Incidence

- Female : male = 2 : 1
- Lower limb : Upper limb = 3 : 1

Types

1- Congenital Lymphedema

⇒ According to age:

1. Lymphedema congenital = at birth. (usually aplasia)
2. Lymphedema precox = at puberty. (usually hypoplasia)
3. Lymphedema tarda = in adult.

⇒ According to finding in development of lymph vs

A. Hypoplasia

- In which the number of lymph vessels is reduced in the affected limb.
- Example in the thigh there one or two vessels instead of the usual five or more.

B. Aplasia: no lymph vessels are demonstrated.

C. Varicose lymphatics: destroyed valve.

- The condition is often associated with incompetent valves.

2- Acquired Lymphedema *It may be due to:*

1. Repeated attacks of streptococcal lymphangitis (i.e. cellulitis).
2. Filariasis (commonest cause).
 - a. W. bancrofti --> in UL. & scrotum.
 - b. W. malyi --> in LL & breast.
3. Irradiation.
4. Surgical excision of LNs which drain the limb (as after radical mastectomy).
5. Obstruction of the lymphatic by malignant tumor.
6. Burns at site of LN.
7. Injuries as circumferential scars of the limbs.



Clinical picture

1. Swelling of arm or legs
2. Heavy: tightness of arm or leg
3. Restricted range of mobility
4. Aching or discomfort of arm or leg
5. Recurrent infection in the affected limb
6. Hardening and thickening of skin and axilla



⇒ Clinical classification of lymphedema (Burner)

Grade	Clinical features (Burner)
Latent	Excess interstitial fluid and histological abnormalities of lymphatics but no clinical lymphedema
I	Edema pits on pressure and swelling disappears on elevation and bed rest
II	Edema does not pit and reduce on elevation
III	Edema is associated with irreversible skin changes, fibrosis, papillae (elephantiasis)

⇒ Complications of Lymphedema:

1- Infection:

- It makes the vulnerable arm or leg more liable to infection including cellulitis, lymphangitis

2- Elephantiasis:

- It occurs when arm or leg becomes so hardened with thickened skin that you have difficulty to move it

3- Lymphangiosarcoma (rare)

- It originates from LN and lymph vessels

Treatment of Lymphedema in General

Treatment is mainly conservative.

A. Palliative:

Indicated in early cases

1. Exercise and manual lymphatic massage
2. Intermittent pneumatic compression
3. Intermittent limb compression pump.
4. Antibiotics for infection (long acting penicillin) : 1,200,000 units every three weeks.
5. Treatment of the cause (Filariasis).
 - Hetrazan.
 - Anti-mosquito measures.

B. Surgical Treatment:

The only indication is disability as results of surgery are not promising.

⇒ Idea:

- Diversion of the lymphatic flow through the deep fascia to lymphatics of the muscles.
1. Swiss-roll cake operation: (Thompson operation)
 - Excess skin is not removed but shaved and buried in muscle-like swiss roll.
 2. Amputation:
 - Indicated if the limb is hugely swollen, ulcerated & infected.

⇒ Other options:

- 1- Charle's operation: total excision of skin, SC tissue, deep fascia then covering using split skin graft.
- 2- Lymphovenous shunt.
- 3- Micro-lymphatic transe operation.
- 4- Omental flap operation.

Filariasis

Cause

- **Filaria bancrofti.**
- It attains sexual maturity in lymphatics → release millions of microfilaria.
- The vector is **Culex pipiens** (culex fatigans).

Pathology

⇒ **4 stages:**

1. Stage of soft pitting edema early.
2. Stage of lymphorrhea due to rupture of lymphatic vessels discharging lymph in subcutaneous tissue.
3. Stage of fibrosis: ↑ protein → excites fibrosis → non-pitting edema.
4. Stage of elephantiasis: thick rough skin from severe fibrosis → pigmentation and ulceration, varicosity of the lymphatics.

Investigations

- **Mid night thick blood film** → to detect microfilaria.

Clinical picture

1. History of the cause:

- Endemic area for filarial.
- Past history of mastectomy or irradiation.
- Recurrent erysipelas.

2. Progressive edema:

- At first, it's a slightly **pitting** edema.
- Latter on, it becomes **non-pitting** due to subcutaneous fibrosis.

3. Skin then becomes thick, rough & dark (Elephantiasis).

- Ulceration with bad odor & toxemia.
- Chylous ascites & hydrocele.
- Chylous fistulae on the scrotum & groin.

4. Elephantoid fever.



Stemmer's sign

- A thickened skin fold at the base of the second toe or second finger that is a diagnostic sign for lymphedema.
- A positive result occurs when this tissue cannot be lifted but can only be grasped as a lump of tissue. In a negative result, it is possible to lift the tissue normally.



DD

(of swollen limb):

⇒ According to the distribution:

A. Localized swelling:

1. Post-phlebotic limb.
2. Inflammation & allergy.

3. AV fistula with local gigantism.
4. Adiposa delerosa (Dercum's disease).

B. Generalized swelling:

- Renal, cardiac hepatic, nutritional, myxedema, Cushing syndrome.

⇒ **According to laterality**

A- Bilateral: causes of generalized edema.

B- Unilateral: causes of peripheral welling.

- 1- Venous edema → DVT.
- 2- Neurofibromatosis elephantosis.
- 3- Lymphatic edema.
- 4- Congenital A-V fistula → local gigantism.
- 5- Huge sarcoma of the limb.

Investigations

1. Lymphangiography → obstruction.
2. Lymphoscintigraphy.
3. Duplex: for venous insufficiency.
4. CT & MRI: better.
5. For the cause e.g. midnight thick blood film for microfilaria.

Treatment

- Anti mosquito measures.
- Hetrazan (diethylcarbamazine citrate).
- Treatment of the streptococcal infection.

Acute Lymphangitis

Definition

- Acute inflammation of lymph vessels secondary to infected wound

Causative Organism

- Streptococcus pyogenes or staphylococcus aureus

Clinical Picture

General ➤ FAHM

Local

- Affected lymphatics appear as tender, hot lines
- Toxemia is often severe
- Suppuration may occur

N.B.:

- Deep lymphangitis is evidenced by general malaise, fever, swelling of affected limb with complete absence of localization

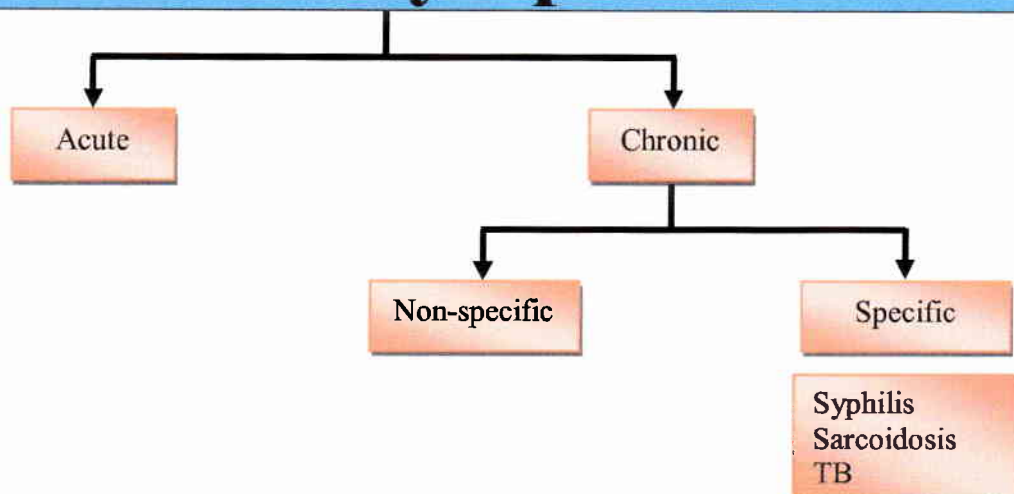
Complications

- Permanent lymphatic damage → recurrent attacks of infection, lymphedema

Treatment

- Bed rest
- Rest of affected limb, elevation
- If suppuration → incision and drainage
- Antibiotics (broad-spectrum)
- Care of primary wound

Diseases of Lymph Nodes



Acute Lymphadenitis

- Acute inflammation of LNs carried to it by lymphatics from area drained by involved gland
- Lymphatics show no evidence of disease except in some strep. Infections

Clinical Picture

- General: FAHM
- Local: painful enlarged LNs with congested edematous, hot overlying skin
- Major cases resolve but suppuration may occur

Treatment

- Antibiotics, local heat, pus drainage if formed

Chronic Non-specific Lymphadenitis

Etiology

- Low grade infection as septic tooth

Clinical Picture

- Enlarged LNs with little pain or tenderness, firm discrete, non-adherent to skin with little tendency to suppuration

Treatment

- Of the primary focus

TB lymphadenitis

Bacteriology

- Caused by human or bovine **Mycobacterium tuberculosis**.
- Stained by **Z.N. stain** because it is acid fast bacilli.
- Cultured upon:
 1. Dorset egg media.
 2. **Lowenstein Jensen media** which is selective for TB bacilli as it contains **Malachite Green**.

▶ **Organism** .
 ▶ **Blood, lymph (Born)**.
 ▶ **Comp.:** abscess + spread
 ↓
 [cold, 2ry inf., sinus, ulcer, pr., calcif]
 ▶ **Invest.:** CBC, PCR, Tuberculin, Biopsy
 ▶ **TTT:** Medical, Surgical

Pathology

- **Tubercles are formed in which there is:**
 1. Epitheloid cells.
 2. Giant cells (Langhan's cells).
 3. Lymphocytes.
 4. Fibroblast.
 5. Caseation
- Depending on the resistance of the patient *caseation & fibrous tissue* is formed.



Histology of LN

- Tuberculous lymphadenitis is common in children & young adults.
- Those who have been in contact with open human tuberculosis or who drink infected milk.
- **TB bacilli might reach the lymph node through the blood (blood born-lymphadenoid type) or through the lymphatics (lymphatic born).**

	Blood borne	Lymphatic borne (fibrocaceous)
Age	More common in elderly.	More common in children.
Origin	Generalized affection. Occurs in milliary TB. OR Blood borne from pulmonary or renal TB.	Localized affection (from <i>catchment area</i>) → Ist complex - Upper deep cervical LNs (the commonest ✓✓) → portal is tonsils (organism pass without reaction) - Mediastinal LNs → from pulmonary TB. - Mesenteric LNs → Intestinal TB
Pathology	Organisms reach L.N. through arterial supply in hilum → affection of medulla - Not affecting capsule (peradenitis) - No matting. - Late caseation. - No cold abscess or sinus is ever seen clinically in this type.	Organisms reach L.N. through afferent lymphatics → affection of cortex - Early affecting capsule (peradenitis) - Early matting. - Early caseation forming cold abscess → perforate deep fascia → Collar stud → Abscess may break down → sinuses

Clinical picture	1- Manifestations of pulmonary TB. 2- Affected LNs → enlarged, not tender, rubbery , not matted & discrete	C/O: → Painless swelling gradual onset, slowly progressive. O/E: 1- Manifestations of T.B. toxemia (2N2L) 2- Manifestations of Pulmonary TB. (dyspnea, cough, expectoration & hemoptysis) 3- Affected L.N. → enlarged, not tender, firm early multiple then matted 4- Cold abscess → fluctuant (But not as hot as pyogenic abscess) 5- Multiple sinuses with undermined edge & cyanotic margins.
DD	a. Hodgkin's lymphoma b. Chronic non specific lymphadenitis	- LNs secondaries For Cold abscess (from other swelling in the neck) For Sinus (from sinuses in neck) thyroglossal – branchial-actinomycosis

- From tonsils the infection spreads by lymphatics to the nearest lymph node i.e. the upper deep cervical LNs → coalesce → cold abscess.
- The caseous material perforates the deep fascia → (**Collar-stud abscess**).
- This skin gradually, breaks down, & forms a sinus.
- Patient usually has tuberculous toxemia.

Complications

1. **Cold abscess formation** which is called so because there is no signs of inflammation (no redness, or hotness).
2. **2nd infection** which is called **hot cold abscess**
3. **Sinus & ulcer** formation.
4. **Pressure** on nearby structure e.g. mediastinal \$.
5. **Calcifications**
6. **Systemic Spread.**

Investigations (as any TB +ve)

1. Blood:

- Anemia.
- **Leucopenia.**
- **Relative lymphocytosis.**
- Increased ESR.

2. Aspiration & examination of caseous material by:

- Naked eye.
- Microscopically.
- Guinea pig inoculation.
- Culture on Dorset egg media for tuberculous bacilli.

3. Smear from sinus & examination for TB bacilli:

4. Lymph node biopsy & culture: better under general anesthesia.



T.B. sinus

For pulmonary TB**5. X-Ray Chest**

- Pulmonary tuberculosis.
- Mediastinal node affection.
- Calcified lymph node.

6. Tuberculin test; it is a good negative test.

7. Sputum analysis: Zeil Nilson stain & culture on Lowenstein Jensen media.

Treatment**a) Tuberculous lymphadenitis before caseation:**

- The general condition of the patient is improved by good diet and nutrition.
- At least two **antituberculous drugs** are prescribed for at least 9 months.
- A combination of rifampicin and isonicotonic acid hydrazide (INH) is very efficient.
- Surgical excision is indicated for a single group of lymph nodes showing no response to medical treatment after a period of 6 months.
- A tuberculous abscess or sinus maybe included with the nodes during excision.

b) Cold abscess:

- Antituberculous drugs
- Aspiration and injection of streptomycin solution. The rules of aspiration of a cold abscess should be followed to avoid sinus formation :
 1. The needle is inserted in a healthy part of the skin away from the abscess.
 2. The site of puncture should be in a non dependant part.
 3. - The needle should also pass in a valvular manner.
 - The points of entry through skin and abscess cavity should not be close and opposite each other.
 - To accomplish this the needle pricks the skin is advanced some distance through the subcutaneous tissue, and is finally thrust deeply to enter abscess cavity.
- **Incision is indicated in the following conditions**
 1. Secondary infection transforming the cold abscess into acute pyogenic abscess.
 2. If the abscess is imminent to rupture , it is incised , curetted , powdered with streptomycin and closed.
- **Excision together with underlying lymph nodes is done if the nodes need excision**

c) Treatment of a tuberculous sinus:

- General antituberculous treatment
- Dressing with streptomycin powder every three days until it closes.
- Excision with underlying nodes, if resistant to conservative measures.

Treatment of Blood borne Type:

- After confirmation of diagnosis by biopsy , antituberculous treatment is prescribed.

Treatment of lymphadenoid Type:

- It is only medical treatment.

Syphilitic Lymphadenitis

Affected in the 3 stage

1-1st stage (chancre):

- a- Genital chancre: → shotty nodes i.e. firm painless, mobile & discrete.
- b- Extra-genital chancre → enlarged, painful, tender, soft (1 w after chancre & disappear 1 m after it)

2- 2nd stage:

Generalized & shotty (sp. epitrochlear & on posterior border of sternomastoid) + condyloma + mucus patches + +ve W.R.

3- Tertiary S (gumma)

- 1- Gumma of L.N. → rare.
- 2- Infected Gumma → acute lymphadenitis.

Lymphomas

- These are **malignant tumors of RES (liver, spleen, BM, blood)**
- The malignant cells are **not present in the blood stream** (localized in the RES) ≠ Leukemia.
- It arises from stem cells (B, T lymphocyte or histiocytes).

Hodgkin's disease

Age & sex

- Sex : It is common in male.
- Age : has 2 age peaks 15-35 and above 50 years old.

Predisposing factor

A. Genetic factors :
P53 gene

B. Infections → EBV

Sites of affection

A. Lymph nodes:

- The cervical (82%)
- The mediastinal (3.5%)
- Abdominal (2%)
- Axillary (9%).

It usually starts in one group & later on it involves other groups of LNs.

B. Spleen, Liver intestine & BM (i.e. extra-nodal lesion)

Macroscopic features

I. LNs:

⇒ **Site:**

- Usually lower deep Cervical → then axillary → then mediastinal
- The large LN is the first to be affected.

⇒ **Character:**

- Enlarged + satellites
- Discrete early → matted later.
- They are rubbery in consistency.

- ▶ Cervical → axillary → mediastinal → abdominal
- ▶ **Staging: 4 stages** (a)&(b)
- ▶ Pleomorphism, Dorothy-Reed, Lost architecture
- ▶ **C/P:** fever, LN., hepatosplenomegaly
- ▶ **Invest.:** Routine, LN biopsy, Staging.
- ▶ **TTT:** Chemotherapy, Radiotherapy

⇒ Cut section:

- Pink in color
- Homogenous with loss of architecture (no differentiation between the cortex & medulla)
- With intact capsule in early cases.
- No caseation.

II. The spleen might be affected: (Toffee - almond appearance).

What are causes of enlarged matted LNs in Hodgkin? (4 after)

1. After time → late case.
2. After irradiation.
3. After biopsy because of the fibrosis.
4. After Infection.

Microscopic

- **Pleomorphism** is the main features (lymphocytes, plasma cells & eosinophils).
- There are characteristic giant cells known as **Dorothy-Reed cells** (Reed-Sternberg cells (as mirror image, overlapped nuclei)).
- The lymph node **architecture is lost**.

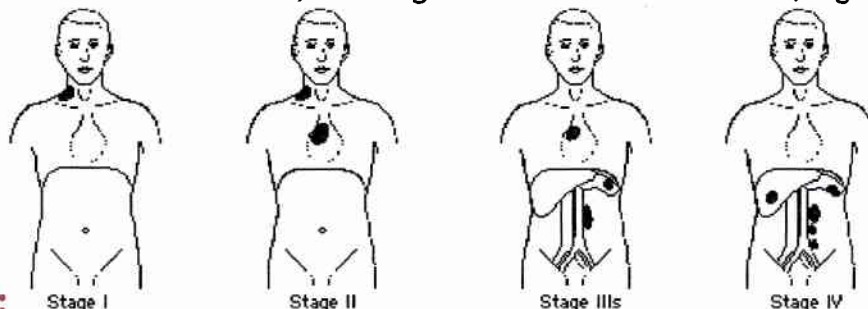


Histo-pathological staging (in descending order of prognosis)

1. Lymphocytic predominance (best prognosis).
2. Nodular sclerosis (with more fibrous tissue) commonest variety
3. Mixed cellularity.
4. Lymphocytic depleted (worst prognosis).

Clinical staging (Ann Arbor classification)

- It is clinically divided into **four stages**.
- Each stage is further subdivided into two groups (a) & (b) according to absence or presence of general symptoms.
- **The general manifestations;** as: weight loss > 10 % in 6 months, night sweats and fever.



⇒ Stage I:

- One group of lymph nodes is affected **OR** Single extranodal site.

⇒ Stage II:

- Two groups of lymph nodes are affected on the same side of diaphragm **OR** Single extranodal site and 1 or more LN areas on same side of diaphragm.

⇒ Stage III:

- One group of lymph node is affected above the diaphragm & another group is affected below it.
- The spleen is enlarged (some consider enlarged spleen as stage IIIs).
- Other consider the spleen to be a group of LNs.

⇒ Stage IV:

- Extra-nodal affection especially liver & bone marrow.

Clinical Picture

Type of patient

Adolescent or middle-aged male complaining of painless enlargement of lower deep cervical LNs

May be associated with:

▪ **3Ps**

- **P**el Ebstein like fever (characteristic intermittent fever which lasts for 2 to 3 weeks followed by remission for few weeks.)
- **P**ruritis.
- **P**ain with alcohol "**Gordon's test**"

☞ **Hepato-Splenomegaly** of unknown etiology in 30% of the cases

☞ **Other manifestations:**

1. **Mediastinal syndrome:** dyspnea, dysphagia, hoarseness & horner's
2. **Jaundice:**
 - a. Obstructive jaundice: due to enlarged LNs in porta hepatis.
 - b. Hepatocellular: due to infiltration of the liver by Hodgkin's tissue
 - c. Hemolytic: due to autoimmune antibodies
3. **Cachexia**
 - Weakness.
 - Asthenia.
 - Anemia.
4. **Metastasis:**
 - Skin metastasis.
 - Bony metastasis.
 - Paraplegia.

☞ **Local examination:**

- **Early:** LN is enlarged, rubbery, not matted etc... (see macroscopic).
- **Late:** LN enlarged matted.

➤ Pain is usually late and with drinking alcohol



Investigation

A) Routine investigations

1. **CBC:** anemia, eosinophilia, ↑ ESR (above 100).
2. **FBS**
3. **CXR:**
 - Mediastinal LN enlarg. appears as wide mediastinum,
 - Paralyzed cupula of the diaphragm (due to phrenic nerve invasion).
 - Rib and/or Lung metastasis, pleural effusion.
4. **ECG**
5. **LFT**
 - Abnormal serum alkaline phosphatase, - Abnormal bilirubin level.
6. **Kidney function tests:**
 - Enlarged abdominal LNs might obstruct the course of the ureter.
 - During ttt excessive tissue destruction → urate stones (**tumor lysis syndrome**).
7. **Bone marrow aspiration:** +ve in stage 4

B) For diagnosisLymph node biopsy (the cornerstone for diagnosis)

- It is better to delay lymph node biopsy for 2 or 3 wks (**Don't rush for lymph node biopsy**) because:
 1. Many cases of lymphadenopathy & fever due to infection; It will improve by time.
 2. The microscopic picture may be extremely **bizarre** early.

C) For staging

1. U/S.
2. Liver biopsy.
3. CT scan
4. Bone scan
5. Spiral CT

6. Staging Laparotomy (now replaced by CT)

- Especially with stage I & stage II
- **The value for laparotomy in Hodgkin's disease**
 - Better staging.
 - Avoid hypersplenism.
 - Protect the kidney from irradiation as we can remove the spleen instead of its irradiation which will affect the kidney.
 - Liver biopsy from both lobes.
 - Abdominal LN biopsy.
 - Putting silver clips on the affected L.Ns. to mark the site for better ttt with irradiation.
 - In females in the child bearing period suture the ovaries behind the uterus to protect them from the irradiation.
 - Appendectomy to see its lymphoid tissue.

➔ **The role of staging is dropping out due to:**

- The high accuracy of CT and MRI
- The risk of overwhelming post splenectomy infection.(OPSI)

Treatment1. **General:** vitamins, blood transfusion, iron therapy.**General scheme for therapy:**

- Radiotherapy for stage Ia, Ib, IIa.
- Stage IIb is treated by radiotherapy and 6 cycles of combination chemotherapy.
- Stage III, IV are treated by 12 cycles of chemotherapy (Mustin, oncovine , procarbazine, prednisone ; **MOPP**) while radiotherapy is supplement to control bulky cervical LNs.

2. **Surgical excision:** is indicated for

1. Biopsy.
2. Laparotomy.
3. Intestinal obstruction.
4. Jaundice.

Prognosis

- Depends upon histological type and grade 5 years survival is obtained in 80% with proper treatment – for stage Ia with lymphocytic predominance results may reach 100%

Non-Hodgkin's Lymphoma

	Hodgkin's disease	Non Hodgkin's disease
Age	Young	Old
Site	Unicentric	Multicentric
Skin overlying	Normal	May be red, hot, & dilated veins
Consistency	Rubbery	Hard
Mobility	Mobile, discrete but late might be amulgamated	Amulgamated early
Tenderness	- ve	- ve
Prognosis	Good	Bad

Classification

Lukes & Collins Classification

it is based on the cells of origin, thus non Hodgkin's lymphoma is divided as

- I- Cell type (Thymus derived: T-cell).
- II- Cell type (bone marrow derived: B-cell).
- III- Cell type (Unclassifiable).

Treatment

(Mainly by chemotherapy as it is multicentric)

As Hodgkin's lymphoma but:

- In nodular type single agent chemotherapy is given (chlorambucil),
- In diffuse type combination chemotherapy is recommended which is cyclophosphamide 400 mg/m², Vincristine 1.4 mg/m², prednisone 40 mg/day.

Burkitt's lymphoma

- Is a highly malignant B-cell tumor that may involve sites other than LNs & RES.
- There is strong evidence that it may be due to EBV & may be related to malaria.
- It is common between ages under 12 years, more in males from eastern Africa.
- Commonly multifocal, affect the jaw (50%) forming painless progressive swelling, ovaries, retroperitoneal tissues & CNS

Histopathology:

Dark blue lymphocytes & starry shaped faint histiocytes (Starry sky appearance).

Treatment:

- by combination chemotherapy & tumor debulking if possible.

